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FACTORS IN RURAL HEALTH IN MINNESOTA

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IT is indeed a pleasure and a privilege to be with you today. This, I believe, is particularly true since one of the objectives of the Farm Bureau is also one of the goals of the Minnesota State Medical Association. This common objective is to see that all of the people of the state, farmers as well as others, are provided adequate high-quality medical care at a cost which they can afford. My remarks today will attempt to portray present conditions of rural health in Minnesota and the efforts being made by the organized medical profession to see that adequate good medical care is available in the near future at low cost.

Before the war, and it is hoped that with military physicians returning to civilian practice, these conditions will soon be repeated; there were 3,600 practicing physicians in Minnesota. Of this number 1,800, approximately, practiced in the urban centers—Minneapolis, St. Paul, Duluth and Rochester. In these centers of population, there were about 1,000,000 of the state's 2,700,000 population. Thus, then, 1,800 physicians provided medical care for 1,700,000 people living in rural districts.

During the war, induction of civilian physicians into the military forces created some maldistribution of civilian physicians. In fact, during the war, there were several instances in which persons living in rural districts had to travel excessive distances for medical care. But before the war, two surveys were conducted, one in 1934 and the second through the press in 1941, to determine whether physicians were readily accessible to all people in Minnesota. Both of these surveys showed that beyond question doctors were available to practically all of the people of the state. It is anticipated that some of the maldistribution of physicians resulting from the war will soon be corrected by the return of military physicians to civilian practice.

The next question that presents itself is, "Under these circumstances, what has been the state of health of the people of Minnesota?" The death rate from all causes has been slightly lower in Minnesota than in the United States as a whole. Life expectancy has increased from forty-nine years at the turn of the century to sixty-one and sixty-five years in 1945 for males and females, respectively. The maternal mortality, newborn and infant mortality rates in Minnesota have been the best of any but one or two states in the Union. The mortality rate from infectious diseases in Minnesota ranks Minnesota among the leading states in public health and preventive medicine. The mortality rate in tuberculosis in Minnesota has decreased to less than one-fourth of what it was in 1910. In fact, in a recent article by Dublin in the *American Magazine*, Minnesota ranked second among all the states of the United States in matters of health.

It appears only reasonable to assume that adequate high-standard medical care is available to all people in Minnesota or the conditions just described could not be true. There are three other factors which are largely responsible for the excellent health of the people of Minnesota. They

Address of the President of the Minnesota State Medical Association before the 27th Annual Meeting of the Minnesota Farm Bureau Federation at the Hotel Lowry, St. Paul, Minnesota, January 14, 1946.

are: (1) the Minnesota Medical School and University Hospital, (2) the Minnesota Department of Health, and (3) the Mayo Clinic at Rochester. Through these three agencies, the very latest measures in diagnosis, treatment and preventive medicine are made available to the general practitioners and the people of the state. All of these institutions have national and even international reputations which have placed Minnesota in the foreground in all fields of medicine.

So, we arrive at the question, "How is the cost of adequate high-standard medical care to be reduced?" So far as hospital care is concerned, the question has been answered in the Blue Cross Hospitalization Plan. Already, approximately 25 per cent of the state's population participates in this plan. Also, it is anticipated that further increases in hospital beds will be available in the not-too-distant future as a result of the recent passage by Congress of the Hill-Burton Hospital Bill. Governor Thye has already appointed a committee of twenty-four members to study the need of additional hospital facilities throughout the entire state of Minnesota. The federal bill provides as much as 100 million dollars a year for the construction of hospitals. What portion of this may be allocated to Minnesota is dependent on the findings of the committee as well as other factors involved.

Prepaid medical care in Minnesota perhaps should be mentioned under three headings, such as: (1) Plans already in operation, (2) state plans, and (3) national plans affecting Minnesota.

Under the first subdivision falls the Farm Security Administration medical care program. Such plans are now in their fifth year. Originally, farm families of the marginally indigent group in Otter Tail, Mille Lacs and Morrison Counties were eligible to participate. At present the plan is operative only in Morrison County. Participation costs $30 per year per family. Families average 5.2 persons. All medical care except non-emergency surgery is provided in the plan. Free choice of physicians is assured the families.

For the first two years the annual cost per family was $23 instead of $30. During this period the number of families eligible for participation dropped about 50 per cent. Also, during this period it was difficult to convince families that they should join. During the past two and one-half years, even with the annual family cost raised to $30, families voluntarily request membership. In fact, the plan has been so successful from the farmer's point of view that many outside families have had to be refused the privilege of participating.

In the second group, state plans first crystallized at the last session of the Minnesota State Legislature. Then, at the instigation of the Minnesota State Medical Association, enabling legislation was passed for the inauguration of a state-wide prepaid medical care plan. Already twenty-four physicians throughout the state have been chosen to serve as organizing directors of the plan. These twenty-four physicians have been organized in four subcommittees to arrange articles of incorporation, organization and administration, contracts and fees and funds necessary for incorporation. Several meetings of these subcommittees have been held. Articles of incorporation have been drafted but not approved. Discussions are being held concerning the coverage of such contracts. That is to say, it is not known at present whether all medical, surgical and confinement services will be included or whether only surgical and confinement cases will be covered. Also, it has not been determined whether administration will be linked with the Blue Cross Hospitalization service.

It is expected that contracts probably can be purchased by anyone. Free choice of physician is assured contract holders. It is hoped and expected that the contracts will be within the financial reach of all who desire to purchase them. It is not known when the plan will become operative, but these facts have been given you to keep you abreast of the plans to date. Suffice it to say that energetic efforts are being made by the Minnesota State Medical Association to see that prepaid medical care of high quality will soon be available to all people in Minnesota.

National plans, the third category, is one factor which is causing some delay with present state plans. The American Medical Association is considering a national prepaid medical care plan which will cover all states. It is anticipated that some time during the month of February more definitive action will be taken on this federal plan. Until it is known whether or not the national plan is soon to be organized it is not entirely clear whether the final steps in organizing the Minnesota plan should be taken. But it is clear that soon, through either one or the other of these developments, prepaid medical care con-

(Continued on Page 258)

TROPICAL DISEASE HAZARDS IN THE NORTHWEST

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TO the average American physician the subject of tropical medicine has a very alien sound. But one of the many things we have learned in recent years is that what is alien is not necessarily unrelated to us. Not long ago Corregidor, Anzio, and Okinawa were strange and curious sounds, but the war brought them to our front door. And as our servicemen and women return they will bring with them medical problems with which we must become acquainted, tropical diseases of which we may hardly have heard, yet which potentially threaten our national health.

In considering tropical medicine, it must be recognized that the term tropical is loosely applied, and not strictly geographic in meaning. Certain tropical diseases, it is true, occur only in the Tropic Zone. But others are more widespread, occurring throughout the world, though more common in the tropics and Orient where climate, dense population, social habits, and poor hygiene provide a more favorable environment for them. Some are even now endemic in the United States, while others have appeared here as epidemics in the past.

The possibility of spread of tropical diseases by returning service personnel depends on a number of factors, of which two are most significant. The first is time. Meleney[22] points out that acute diseases with short periods of communicability such as yellow fever, typhus, plague, and cholera, develop and run their course quickly; hence, if encountered at all, will be dealt with while the veteran is still en route home, or before discharge. They therefore have little chance of establishment in the United States and civilian doctors run even less chance of meeting them. In contrast are those diseases which are chronic, with a long period of incubation or communicability; hence, a chance to bridge the period of demobilization. Such are malaria, the dysenteries, and the helminth infestations. These we will undoubtedly see to a greater or lesser degree.

A second factor determining hazard of introduction or spread of a particular disease is means of transmission. Some are bacterial in origin and transmitted directly. Some are carried by insects or require presence of other animals as intermediate hosts. Such diseases can be brought back by the returning veteran, but can be transmitted to others only if the proper insect vector and intermediate host occur here.

Practically, therefore, the physician may expect two types of tropical disease problems in his practice. The first may be illustrated by bacillary dysentery, a bacterial infection, or amebiasis, a protozoal disease; either of these may be communicated by direct fecal contamination, or indirectly by the housefly. Such diseases may be seen either in the returning veteran or in the civilian whom he contacts. The second type will be seen only in the veteran. It is well illustrated by African sleeping sickness or trypanosomiasis, which requires the tsetse fly for transmission. This insect is not native to America, and its introduction is not likely. It is quite probable that an occasional serviceman from Africa may carry the disease home unrecognized, and thus come to the physician for diagnosis. But passage of the disease to others is and will remain extremely unlikely, if not impossible. We need not look for it in those who have not been in Africa. This brings out a significant point in dealing with all exotic diseases: ask where the patient has recently been.

Malaria

Turning now from general principles to specific diseases, authorities agree that of all hazards to be anticipated, the chief is malaria. Branch[2] and Getting[14] emphasize that it is the most common, widespread, and lethal disease of man. Throughout the world it is estimated to kill over three million annually. "After disembarkation and demobilization, all previously malaria-free individuals will cease prophylactic treatment. Any change in living may bring out the disease. Already we are having deaths from such disease treated too little and too late." Surgeon General Parran[25] says, "Outside of mental diseases, there is no other disease of comparable importance against which we have made less progress during my generation." Malaria in varying degree is present in most of the countries to which our forces have gone—Africa, the Mediterranean basin, Asia, South Pacific, East Indies, and South America.

As to etiology, the parasitic plasmodia are carried by various species of Anopheles, several of which occur in the United States. The only one of significance in this area, however is A. quadrimaculatus. This mosquito is fairly common throughout southern Minnesota, the eastern Dakotas, and on east and south to the Atlantic border. It is well known that malaria was endemic in much of this region in years past, but modern screening, better mosquito control, improved care of the malaria case, and perhaps climatic factors inimical to the parasite have made the disease relatively uncommon. Returning service personnel will undoubtedly serve as a pool of malaria infection which may again establish the disease for a period of years unless a well-planned defense is provided by State or Federal public health authorities. The possibility of introduction of some other mosquito vector hitherto unknown in this region is also to be noted, particularly in this era of air transport. Anopheles gambiae was thus introduced into Brazil from Africa some years ago leading to a disastrous epidemic. Public health authorities moved in and finally eliminated the mosquito by a classic anti-mosquito campaign, but only after a program lasting two to three years and costing two and one-half million dollars.

In considering malaria clinically, one must not forget its pathology. The parasites involve primarily the reticulo-endothelial system, leading to splenomegaly and anemia. In addition, there may be liver enlargement and parasitic invasion of capillaries of the brain, meninges, lungs, and intestine, causing cerebral and other complications. Renal damage may be revealed as blackwater fever, due to acid hematin deposits in urinary tubules. The classic picture of remittent fever occurring at forty-eight- or seventy-two-hour intervals is well known. It must be understood, however, that such nice regularity is not to be expected in every case, or even the average case, particularly in this region. Any unexplained fever, no matter whether regular or irregular, marked anemia, splenomegaly, recurrent diarrhea, or sudden, severe cerebral manifestations require immediate and intensive blood smear examination for parasites, if the patient has formerly been in an endemic malaria region. Diagnosis must be based on positive blood findings; smears should be prepared by both thin and thick film techniques, and should be repeated on consecutive days if initially negative.

The laboratory differentiation of the several malaria parasites is not merely of academic interest. It is of definite importance in treatment and prognosis. It is generally agreed that quartan and ovale forms are not to be expected commonly in the United States. Differentiation will, therefore, lie chiefly between the vivax and falciparum or aestivoautumnal strains. All authorities agree that falciparum malaria is the more vicious type, with parasites sometimes invading up to 5 per cent of the red corpuscles, leading to severe cerebral and meningitic manifestations and the dreaded blackwater fever. On the other hand, Butler and Sapero[5] point out that as seen in the South Pacific, falciparum malaria has relatively low inclination to latency, with a recurrence rate of only 5 per cent; hence it is less likely to be carried into the United States. Also as Meleney[23] notes, it is more amenable to suppression by prophylactic use of atabrine.

The commoner tertian or vivax malaria as found in the South Pacific is said, on the other hand, to be very chronic, resistant to atabrine, tending to recurrence over a long period of time. It is two to three times as apt to recur as our domestic strain of tertian malaria, and may be expected to continue in a patient for an average of two or more years.

The treatment of malaria has had interesting developments as a result of the war. For example, Talbot[31] reports on two groups of men in the South Pacific endemic area. An army group was given routine suppressive doses of atabrine. A navy group in the same area was treated only as clinical malaria appeared. Later a survey of the two groups showed that 48 per cent of the army men had latent malaria as shown by positive blood smears, as compared with 66 per cent of positives in the untreated navy group. Further, clinical malaria developing in the army group during suppressive treatment tended to be more severe than in the untreated navy men. This and other similar experiences have led to the belief that where proper laboratory facilities exist, it is better to omit suppressive therapy even in heavily endemic areas, and treat only active clinical cases.

Routine treatment is thoroughly covered in the Surgeon General's Circular Letter No. 153.[29] It is here emphasized that the plasma level of antimalarial drugs is fundamental in determining

their efficiency. Of the two commonly used drugs, quinine localizes less in the tissues and so acts sooner, while atabrine first saturates the tissues and thus acts longer but more slowly. For suppressive therapy, advisable only when troops must be inadequately protected in heavily endemic regions, the recommended dosage is: atabrine 0.1 gm. at the evening meal 6 days a week, or quinine 0.6 gm. daily at the evening meal. For proper suppression the drug must be taken regularly.

For clinical treatment of the active uncomplicated case, recommended therapy is: atabrine 0.2 gm. with 1 gm. sodium bicarbonate in 200 or 300 c.c. of water or fruit juice every six hours for five doses, then 0.1 gm. t. i. d. p. c. for six days; or quinine may be given, 1 gm. t. i. d. p. c. for two days, then 0.6 gm. t. i. d. p. c. for five days. In falciparum cases, to control the gametocyte or sexual stage, plasmochin may be necessary, though it is a toxic drug and must be used with caution; give it after a course of atabrine, or along with quinine, 0.01 gm. three times daily for four days, with 1 gm. sodium bicarbonate per dose. For severe cases, or those with complications of vomiting or coma, atabrine is preferred, in doses of 0.2 gm. in 5 c.c. of sterile distilled water intramuscularly in each buttock, repeated one or two times at intervals of six to eight hours if necessary. Turn to oral medication as soon as possible such that total dosage is 1 gm. in forty-eight hours, then 0.1 gm. t. i. d. p. c. for five days; or quinine may be given, 0.6 gm. in 300 to 400 c.c. sterile isotonic salt solution intravenously, repeating if necessary, and following with atabrine or quinine by mouth as soon as possible. Treat relapses the same as first attacks. If relapse is suspected, but blood smears negative, parasites may usually be demonstrated after giving epinephrin, 0.5 c.c. of 1/1000 solution subcutaneously. In all malaria treatment, possibility of quinine or atabrine sensitivity must be kept in mind, in which case the other drug must be relied on alone. Totaquine, the purified total alkaloids of cinchona, may be used as a substitute for quinine, in equal dose.

Helminth Infestations

Turning now to worm diseases, the commonest throughout battle areas has presumably been ascariasis, due to the roundworm often found in the United States. The life cycle of this worm involves passage of eggs in the stool, contamination of water or food, particularly vegetables or fruits, and re-entrance to the human intestinal tract. Here the egg hatches, the larva penetrates intestinal capillaries, is carried to the lung, penetrates the alveolar wall, is coughed up to the larynx, reswallowed, and develops into the adult worm, ready to produce more eggs. Preferred treatment is hexylresorcinol (coated pills) 1 gm. on an empty stomach, followed in an hour by a saline cathartic. This is also effective against pinworms and whipworms.

Hookworms are fairly common in the more cultivated wet areas in the Pacific, but the disease picture is sufficiently well known in this country not to require special discussion here.

Filariasis, a roundworm infestation of different type, has appeared rather frequently among troops in certain South Pacific regions. By its very nature, causing tissue swelling, edema of the lower extremities and genitalia, and sometimes elephantiasis, it has been much feared by servicemen and many physicians. The disease has existed for many decades as an endemic focus in Charleston, South Carolina, to which it was brought by the slave trade. Due to natural conditions unfavorable to the parasite, the disease has almost disappeared there. As seen in the Pacific, the complaint is prevalent in rather sharply limited areas where dense population and presence of a suitable mosquito carrier have made a fertile field for the infection when once introduced. The cycle begins with sucking up of microfilariae or young parasites by a mosquito, Culex fatigans or other suitable vector; the parasites invade the mosquito thorax muscles, migrate to the proboscis as larvae, and are re-deposited in man, find their way to lymphatics, and there mature, producing their microfilarial young. The worm, called Wuchereria bancrofti, is hair-like in diameter, 2 to 3 inches long, and by preference locates in lymphatics of the groin and genitalia. Clinical symptoms appear only when parasites are introduced in considerable numbers by frequent mosquito attacks. Troops soon moved from an endemic area apparently suffer little damage from an early infection. Even though considerable swelling has appeared, it is noted that in nearly all cases the lymphedema quickly and permanently disappears on return to a temperate climate. Diagnosis of early cases may usually be made by finding enlarged epitrochlear glands and a rubbery swelling of the spermatic cord. An intradermal diagnostic test using an antigen prepared

from Dirofilaria immitis[32] has been reported valuable in detecting early suspects. Holmes[17] recommends excision of enlarged nodes when troublesome. No other specific therapy would appear necessary in areas where proper mosquito carriers do not occur, but where more aggressive therapy appears indicated, lithium antimony thiomalate, as recommended by Brown,[3] would appear promising. Stibamine glucoside[10] ("neostam"), useful in kalaazar, has also been suggested.

Onchocerciasis and loa loa, causing fugitive subcutaneous swellings, may possibly be seen in returning troops, and will be of clinical significance, we are told, chiefly when involving the eye, causing conjunctival swellings and occasionally blindness. Dracontiasis or Guinea worm may perhaps occur as subcutaneous swellings and blisters of the legs and feet. Local treatment may be necessary. None of these maladies can be expected to spread in this country.

Tapeworm and echinococcus infections may be dismissed as unlikely hazards in the United States, resulting only from the use of grossly contaminated water or food, a point strictly watched by the army sanitation authorities.

Blood Flukes

Diseases resulting from infection by various flukes are, in contrast, expected to appear in varying degree. Several types, Fasciola hepatica, Chlonorchis sinensis and others, are unlikely, requiring as they do the eating of uncooked fish, etc. Real hazard exists in the case of the schistosome group, however; the eggs of these parasites are passed in the stools or urine, develop in fresh water into ciliated larvae, invade certain species of snail, re-emerge into the water, and penetrate the skin of man when he wades or bathes there. They proceed to the capillaries of the intestine, urinary bladder, or lung, and there mature, and from those organs the eggs enter the stool or urine. Due to the nature of war, with land troops frequently subject to immersion in contaminated pools and streams, it appears unavoidable that infections must occur in endemic areas. Schistosoma hematobium heavily infests North Africa and to a less extent other parts of Africa and the Near East. Clinically it involves chiefly the venous plexus about the urinary bladder, causing bladder wall abscesses, urgency, pain, and hematuria. S. mansoni occurs widely in Africa and northern South America; S. japonicum is found in Japan, Central China and the Philippines. Both these diseases center mainly in the intestine, with dysenteric symptoms and bowel ulceration, but secondarily may show marked splenomegaly and hepatomegaly. Diagnosis of all forms is through isolation of the parasitic eggs. Treatment is specific, using trivalent organic or inorganic antimony, of which the best is neoantimosan (fouadin) intramuscularly or intravenously. Emetine is also effective, but must be used with caution.

So far as is known at present, schistosomiasis cannot be spread in the United States due to absence of suitable intermediate hosts, certain freshwater snails. However, it is possible that facultative hosts may develop among our own snail species; investigations are now being carried out along this line. Certainly the disease should be watched for among returning veterans, for if present, it is of serious import.

The Dysenteries

The chief advance in the subject of the dysenteries recently has been in the classification and therapy of bacillary dysentery. Callender[6] and others, advocate that this term be broadened to include not only the Shigella dysenteries of Shiga, Flexner, Sonne, et cetera, but also the severe diarrheas of the Salmonella group. It has also been suggested that the Shigella dysenteries be called Shigellosis, paralleling the term brucellosis. All of the intestinal bacillary infections are common under war conditions, particularly in the Orient, and have occurred numerous times among service personnel. Differential diagnosis clinically is most accurately done through careful stool examination, including culture on special media. However, careful epidemiologic analysis will also point to a diagnosis in many instances. Thus an epidemic of food poisoning appears within a very few hours after the ingestion of the causative food, is violent, but should quickly clear under proper treatment. Salmonella infections show up as epidemics, but with an incubation of hours or days, with fairly acute fever and general constitutional symptoms, subside more gradually. Shigellosis varies much more in incubation interval, from days to weeks, shows less in the way of fever and general symptoms, but causes more serious bowel inflammation, with ulceration, bloody stools and tenesmus, continuing over a longer period of time. Amebiasis occurs as a much more

sporadic disease, varying from very mild to severe, and from acute to extremely chronic. For cases particularly difficult to diagnose, the taking of swabs by the technique of Watt and Hardy[16] may help.

Therapy of the bacillary dysenteries[4,12] should begin with 2 to 4 drams of saturated sodium sulphate solution; after two hours give sulfaguanidine or sulfasuxidine 5 gms. q.i.d., or sulfadiazine or sulfathiazole, 1 gm. q.i.d. for two days, then t.i.d. until the stools are normal for two to three days. It is well to check by daily stool culture until two or more daily cultures are negative. Some authors prefer sulfaguanidine, while others prefer the soluble drugs, particularly sulfadiazine.

Though affecting many of our troops while abroad, bacillary infections run too short a course and are too well controlled by adequate epidemiologic measures to threaten serious epidemic spread in this country. All of the infections mentioned are already found here frequently as a matter of fact, and such sporadic outbreaks may be expected to continue.

Amebiasis has been the subject of numerous surveys in the United States, with reports of incidence up to 15 per cent or more. The great majority of cases are silent or minimal symptomatically, hence, seldom secure treatment. Acute, fulminating cases may occur, however, as well as the less common hepatitis and liver abscesses. Diagnosis in the acute case through identification of motile amebae in a warm, fresh stool is a relatively simple procedure. For demonstration of cysts in the chronic case or carrier, rectal swabs may be necessary, with staining by special methods of Faust and D'Antoni.[13] Treatment of the acute case of dysentery or liver amebiasis should begin with emetine hydrochloride, gr. i hypodermically for six days or less. Emetine is a potent myocardial poison and administration must be guarded. In chronic cases, and following emetine in acute cases, any one of several preparations may be used. Carbarsone, an arsenical, is given twice daily for ten days, and in more critically ill patients is given in enemas. Chiniofon (yatren) is an iodine derivative much used formerly, but less in favor as other preparations have been found. Diodoquin, 1.5 to 2 gm. b.i.d. for ten days, or vioform, 0.25 gm. t.i.d. for ten days are also recommended, particularly in subsequent courses of treatment in relapse cases.

Bacterial Diseases

Of the bacterial infections, leptospirosis (Weil's disease, infectious jaundice) is so widespread as hardly to fall in the tropic disease list. It is endemic in Japan. The spirillum is carried by rodents and excreted in their urine. In man the chief effects are swelling and destructive changes in the liver, spleen, kidneys, and lymph nodes. Onset is acute, with fever, headache, nausea, vomiting, diarrhea, and jaundice. Organisms are found in the blood early, and later in the urine for as long as 100 days. An agglutination test and guinea-pig inoculation aid in diagnosis. The possibility of this disease spreading in the United States is undoubtedly present, but uncertain in degree. Treatment is primarily a nursing problem, but may be aided by a specific antiserum.

Relapsing fever, a related disease of wide distribution, occurs in modified form in southwestern states and Central America. It is transmitted by lice or ticks parasitic on small rodents. Clinically the disease resembles the preceding, but icterus and renal changes are less intense and the fever has a definite periodicity, with free intervals of a week or more. Diagnosis is confirmed by finding the causal spirochete in the blood. Treatment by 1 or 2 doses of neoarsphenamine is specific and effective, but should be delayed when a fever crisis is impending. Both relapsing fever and leptospirosis should be subject to epidemiologic control through elimination of the insect vectors in a threatened area with the new DDT preparations.

Yaws, also called framboesia, is a skin disease with constitutional manifestations, caused by a treponema carried by direct contact or flies. It is closely related to syphilis in that it gives a positive Wassermann reaction, is prevented by a previous attack of syphilis, and has marked clinical similarities. The primary lesion usually occurs on the lower extremities, with local adenopathy. There is a secondary septicemia with general adenopathy and fine desquamating papules lasting several weeks. The later tertiary lesions are destructive, with deep scars and deformity; ulcerative rhinopharyngitis is a common development. Organisms should be demonstrable in the primary lesion. Treatment is with neoarsphenamine plus bismuth or carbarsone. Appearance of this disease in the veteran returning from Africa, the East Indies, and the Pacific Islands is presum-

ably possible. Medical standards in this country should prevent any significant spread.

Plague, whether of the bubonic or pneumonic form, should not occur through importation by military personnel. In this connection, however, physicians in this region should perhaps be more aware of endemic plague which occurs among thirty-two or more species of wild rodents in our northwest states, as far east as North Dakota. Human cases have thus far been few and sporadic. When recognized in time, treatment by sulfathiazole has proved quite effective.

Cholera should be mentioned here for sake of completeness, but its introduction into the United States by military personnel is not to be expected. The violence of the infection and brevity of incubation period would surely bring out the infection before the patient disembarked. It is of interest that sulfaguanidine is reported by Huang[18] to have given twenty-one cures in twenty-two patients whom he treated.

Leishmaniasis

Under the term leishmaniasis are grouped three diseases caused by protozoa of special type. Leishmania donovani and one or more related forms are transmitted by species of Phlebotomus or sand fly, and give rise to kala-azar or visceral leishmaniasis. The parasites invade the reticuloendothelial system, causing splenomegaly, hepatomegaly, lymphadenopathy, and occasionally, ulcers of the skin and intestinal tract. The incubation period is usually two to three months. Onset is slow, with irregular fever, visceral enlargement, dark skin, and tendency to dysentery. Blood examination shows anemia, leukopenia, and perhaps the diagnostic parasites, Donovan bodies. Splenic and liver puncture may be resorted to for diagnosis in questionable cases. Therapy is by means of antimony compounds, of which the preferred form is neostibosan or neostam, given intravenously or intramuscularly in graduated doses, one course of ten injections usually sufficing. The disease occurs endemically but fairly widely in Asia, Africa, the Mediterranean basis, and South America. Infection of service personnel may occur, but transmission within the United States is very unlikely as sand flies occur only rarely; they apparently are not found in the Northwest.

Cutaneous leishmaniasis or oriental sore is a maculo-papular, ulcerative, occasionally tuberous skin disease found around the Mediterranean, in India, and South America. Incubation may be as long as three months, and the cutaneous lesions may remain active for over a year. Diagnosis is through finding of intracellular Donovan bodies in ulcer scrapings or biopsy. Treatment is by antimony compounds in smaller doses than those required for kala-azar. Due to its site and nature, this form of leishmaniasis will probably have prompt attention from the military or home physician. In the absence of the sand fly, the disease may be ignored as an epidemic hazard in this country.

Leishmaniasis americana or espundia is limited to Central and South America, and again the sand fly is its carrier. The lesions are flat papules progressing to deep ulceration on the mucocutaneous areas about the mouth, nose, and throat, similar in chronicity to oriental sore. Diagnosis is established by finding the Donovan bodies in ulcer margins. Therapy is satisfactory by use of intravenous antimony tartrate or fouadin.

Trypanosomiasis

Trypanosomiasis is due to protozoan parasites which infest many of the domestic and game animals where they occur. African trypanosomiasis or sleeping sickness has two forms, the West African and East African, due to different parasites, but both transmitted by the tsetse fly. The disease begins with a red nodule termed a trypanosome chancre, which subsides after a few days. The second stage is marked by fever, erythema, malaise, anemia, headache, and lymphadenopathy which particularly involves the posterior cervicals. This stage may last for an indefinite period and may end in death. Most cases eventually go on to a third stage of marked weakness, asthenia, shuffling gait and stupor, the true sleeping sickness. This proceeds to certain death. Diagnosis rests on finding the causative organism in the peripheral blood, or later on in the lymph nodes or cerebrospinal fluid. Early cases respond well to therapy with Bayer 205, given intravenously. For later cases in which the nervous system is affected, tryparsamide must be used; this, however, is effective only in the West African form. In the East African type the disease proceeds more rapidly, and death usually occurs within a year.

Authorities agree that sporadic cases of African trypanosomiasis may occur among American servicemen, and due to the slow course of the disease, may appear after return to the States. The

disease must, therefore, be kept in mind, in view of the grave prognosis if undiagnosed and untreated early by specific agents. Spread of the disease in this country would appear impossible in the absence of the tsetse fly.

American trypanosomiasis or Chagas' disease occurs in South America and Central America, where it is parasitic in rodents and other small animals. It is transmitted to man by triatomas, assassin bugs. The initial lesion is often on the eyelid, with edema and conjunctivitis. Trypanosomes appear in the circulating blood, but assume leishmania forms in the muscles. Myocarditis, brain inflammation, visceral cloudy swelling and lymphadenopathy result. The acute stage is marked by fever, edema, splenomegaly, cardiac weakness and arrythmias, and perhaps meningoencephalitis. The chronic stage is characterized by general visceral enlargement, lymphadenopathy, and irregular fever, with all symptoms gradually diminishing. Death often comes through heart failure. Diagnosis may be established by identifying parasites in circulating blood or smears of lymph gland pulp. Guinea pig inoculation may be necessary. No direct method of treatment has proven effective, but Bayer 205 is reported on favorably. Attacks aimed at the assasin bugs may prove worth while in endemic areas. Like the preceding diseases, American trypanosomiasis may perhaps appear occasionally in service personnel, but transmission within the United States would appear impossible except for the southwestern states, where the assassin bug is advancing from Mexico.

Rickettsial Diseases

Of the rickettsial infections, only two need attention here. Typhus, widespread in the world and prone to increase under wartime conditions, occurs in two forms. The epidemic form is carried by the body louse, infection apparently taking place by crushing of louse tissues or feces into abrasians, rather than through bites. Following a brief incubation period there is onset of sudden, severe headaches, fever, stupor, conjunctivitis, vomiting; later a rash appears, and still later coma, prostration, hemorrhages, ending often in death in twelve to fourteen days by bronchopneumonia, cardiac failure and exhaustion.

The endemic form is transmitted by the rat flea, and due to a slightly different parasite. The disease is less virulent than the epidemic form. Both types may be confirmed in the laboratory by agglutination of X^{19} strain of Bacillus proteus (Weil-Felix reaction). Active treatment of typhus is largely supportive and non-specific. Effective prevention, however, is provided by Weigl's vaccine, a glycerine suspension of infected lice intestines, or by a vaccine prepared in an egg-yolk medium. Due to the short incubation period, the violence of the disease, et cetera, it would appear nearly impossible for any significant number of typhus cases to reach this country in returning armed forces. Furthermore, with sanitary standards existing in the United States, and the potency of our newer insecticide powders, the possibility of a typhus epidemic seems out of the question.

However, a related infection appears of more concern to our present service forces. This is tsutsugamushi fever, or Japanese river fever, a rickettsial infection carried by certain mites in Japan, Korea, and Formosa. A variation of this, called scrub fever, is common in many of the South Pacific islands, and has laid low a fair number of our troops in endemic areas. A small necrotic ulcer initiates the infection, followed by lymphadenopathy, lymphangitis, an eruption lasting six or seven days, bronchial symptoms, muscle and joint pains, prolonged intermittent fever, and often delirium. Fatality rate is said to be about 15 per cent. The disease can be identified by agglutination of the OXK strain of B. proteus. Like epidemic typhus, treatment is symptomatic and supportive only. No preventive therapy has been announced.

Virus Infections

Coming to virus diseases, two seem worthy of consideration. Yellow fever, a severe epidemic and endemic infection carried by various mosquitoes, particularly Aedes egypti, is widespread in western Africa and certain parts of Central and South America. It has invaded North America more than once. In the human body the virus attacks the capillary lining, with virusemia and visceral necrosis. The clinical picture is dominated by fever, jaundice, slow pulse, hemorrhages, and severe kidney damage. Death occurs in 10 to 60 per cent between the sixth and tenth days. Diagnosis can be proven by a mouse protection test. There is no specific treatment of the active case, but excellent protection is provided by vaccination with an avirulent neurotropic virus which is

good for four years. Anti-mosquito measures in endemic regions and quarantine procedures in nearby ports have proved of value. Due to fairly rigid observance of such methods, it is believed that yellow fever should not further threaten North America.

Dengue, breakbone fever, is another virus infection carried by Aedes; it occurs in the Pacific, Near East, South America, West Indies, and occasionally in southeastern United States. After a short incubation period the patient complains of joint pain, headache, erythema, fever, and severe general malaise. There is a brief remission, then a return of symptoms, with skin rash, desquamation, and leukopenia. Diagnosis is presumptive, based on epidemiologic factors. Treatment is symptomatic. No preventive is thus far available. Though troops in the South Pacific have suffered dengue epidemics at various places, transmission in this country would generally be impossible due to absence of Aedes.

A number of other tropical maladies—leprosy, sprue, the less common virus, rickettsial, and parasitic diseases might be discussed here, but do not appear pertinent to the subject as not posing significant hazard to this region.

Conclusion

The accompanying table summarizes the likely tropical disease hazards in the Northwest.

Disease	*Hazard in Returned War Veteran*	*Hazard in Others*
Malaria, Tertian (Vivax)	††	†
Aestivoautumnal (Falciparum)	†	?
Quartan and Ovale	?	0
Ascariasis	†	?
Hookworm	†	0
Filariasis (Wuchereria Bancrofti)	†	0
Other types	†	0
Tapeworm	?	0
Echinococcus Cyst	?	0
Schistosomiasis, Urinary or Intestinal	†	??
Other Fluke Diseases	?	0
Bacilliary Dysentery	†	†
Amebiasis	†	†
Leptospirosis	†	?
Relapsing Fever	?	0
Yaws	†	?
Plague	0	0
Cholera	0	0
Leishmaniasis	†	0
Trypanosomiasis	†	0
Typhus, Epidemic	0	0
Endemic	?	?
Tsutsugamushi Fever, Scrub Fever	0	0
Yellow Fever	0	0
Dengue	0	0

Two general points of caution mentioned by Guldseth[15] and various writers should be noted in dealing with patients who have been in tropical and oriental countries. The first is that malaria, yaws, and a number of other tropical infections may cause positive Wassermann, Kahn, and Kline reactions, leading the careless diagnostician to a wrong conclusion. The second is that returning personnel must be carefully checked by history and blood smears before use as blood donors, if accidental transmission of tropical disease parasites is to be avoided.

In general, the practicing physician or surgeon can prepare himself in four ways to meet the tropical disease threat in the postwar years. First, he should know what diseases are likely to or possibly can appear. Second, he should learn the habit of including tropical diseases in his bedside diagnosis, and not merely leave them as abstract concepts on his bookshelves. Third, he might well see that his laboratory technicians are alive to the special studies so essential in this line of work. And fourth, he might determine where special consultants can be found if need should arise.

In this connection, it is of interest to note that medical educators are aware of the need for wider knowledge of tropical medicine. There are plans for broader courses in a number of medical centers, and for the formation of some type of consultant bureau. Further, concentrated courses have been and are being given to selected groups of physicians under the auspices of the American Association of Medical Colleges, the Co-ordinator of Inter-American Affairs, the Surgeon General, and other agencies, the schedule including a period of field work in Central or South America.

Thus, there is hope that America's wider ventures in international relations may bring new health to other peoples, while maintaining reasonable safety for our own.

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(Continued on Page 294)

FACIAL INJURIES

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THE two immediate concerns in a facial injury are the maintenance of the airway and the control of bleeding.

The tongue must be maintained in its forward position by placing the patient face down. If necessary direct traction is made on the tongue. This is rarely necessary in a conscious patient, except when the symphysis of the mandible and the floor of the mouth are badly disrupted. Bleeding can usually be controlled by pack and manual pressure.

If a trachectomy appears to be needed it should not be postponed until it becomes a desperate and hurried measure. It should be done in an orderly manner under local anesthesia with the trachael opening placed well below the level of the cricoid cartilage. An anxious and hurried tracheotomy is necessary more often because of the surgeon's procrastination than because of immediate urgency at the moment of the injury. When the decision is in doubt one should lean in the direction of commission and create a tracheal opening. It provides an assured airway and a fine means of pulmonary drainage and the patient has a tranquil postoperative course which diminishes the need for sedation and assures one of the patient's co-operation in combating atelectasis.

Shock is not a common feature of facial injuries unless they be extensive and involve vessels of good size. Plasma is a useful immediate measure for combating shock when it exists but the blood volume should be restored at the first opportunity by whole blood transfusions.

It is not unusual in extensive injuries of the face and jaws to have associated cranial involvement. After establishing an airway and restoring the patient's circulatory efficiency one should therefore perform a neurological examination and establish the presence or absence of intracranial involvement.

In severe smashes of the central third of the face which occur as a result of the forward motion of the face against some immovable part of a vehicle or airplane a compression fracture of a vertebra is a common associated injury. It is well to bear this fact in mind when one examines the rest of the skeletal system for injuries.

The facial skeleton when considered surgically can well be divided into two main portions: the mandible, and the bones comprising the middle third of the face.

One has two objectives in the care of a fractured mandible. The first is to get bony union and the second is to obtain good dental occlusion. To accomplish these one sets the teeth of the mandible into proper occlusion with the teeth of the maxilla and fixes them there. The means of fixation vary with different oral surgeons but so long as the fixation is firm and the position is good the results tend to be satisfactory.

There are certain fractures of the mandible that deserve special consideration. Of these, the first perhaps is a fracture of the condyle of the mandible. In general, because of the inaccessibility of the condylar portion of the mandible it is impossible to restore its alignment with the main body. An accepted method of treatment of such fractures is simple splinting of the mandible during the period of pain and swelling. Splinting is done in proper dental position by occluding the lower teeth to those of the maxillae. After the initial period of pain has passed motion is encouraged and nonunion at the fracture site is established. A method of open reduction to re-establish proper alignment of these fractures may be used, but the first and simpler method has seemed adequate.

Since the teeth are the means of both fixing and positioning the mandibular fragments, a special problem arises when a posterior mandibular fragment with muscular attachments is separated from the remainder of the tooth-bearing portion of the mandible by a fracture line posterior to the last tooth. This fragment, often referred to as the edentulous posterior fragment, is comprised of the vertical ramus of the mandible and any of the horizontal portion extending forward to the fracture site. There are different means of fixing this fragment in position during the

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healing period. None of the means is applicable in all cases. In some cases a controlling wire is inserted at the posterior margin of the vertical portion of the mandibular ramus and is let through the skin posteriorly beneath the lobe of the ear to a hook descending from a plaster head cap. In others a small half-round bar is fashioned and secured to the arch of the lower teeth. A posterior extension of this bar can be bent and angled so that its sharp terminal end can be set into the posterior fragment of the mandible much in the manner of a skeletal pin, thus maintaining the relation to the anterior fragment. It is in cases of the edentulous posterior fragment that external skeletal fixation has its widest application. Paired pins are inserted into the mandible anterior and posterior to the fracture line. The couplets are then connected across the fracture site by a firm bar. In using this technique we have not given the mandible free range during its entire healing period. During the first weeks when union is being established we have assisted the external fixation in its support of the fracture line by fixing the mandible to the maxilla by inter-maxillary wires.

A third major problem in mandibular fractures arises when there is considerable loss of substance of the bone and re-establishment of full continuity and alignment is not possible by simple means. This is the type of mandibular injury often seen in gun shot or shell fragment wounds. Large sections of the mandible are extensively comminuted and fragmented and pieces are displayed into the soft tissues of the floor of the mouth or even down into the soft tissues of the neck. When such injury occurs in the symphysis area and the fragments are of considerable size and driven posteriorly into the muscles of the tongue it is possible to retrieve them by direct traction on the fragments by means of small wire loops inserted through dental drill holes, and attached with elastics to an anterior bar replacing the former position of the mandible. This bar is supported in its place by cementing it firmly to the most anterior viable teeth on each side of the mandibular stumps. Rubber band traction applied over a period of several days will often draw forward the fragments with their muscular and periosteal attachments into a semblance of order in the anterior symphysis area. In the region of the horizontal ramus where this extensive comminution occurs from a penetrating type of wound, it is surprising how frequently the multiple fragments displaced a considerable distance from their proper location will, over a period of several days, due to the retraction of periosteal attachments and shreds which have extended out into the soft tissues with the bone fragments, replace themselves into their original alignment. It is generally impossible to restore these multiple fragments to their original position by manual traction and one must be extremely careful in handling the fragments that the single remaining periosteal attachment is not destroyed. Often only a single nutrient vessel traverses the attachment and means the difference between a live bone fragment and a squestrum. The portions of mandible on both sides of a large area of loss such as has just been described must both individually be supported in proper relation to the teeth of the maxillae. Fixation must be maintained for many months to give the periosteal shreds and the remaining bone fragments full opportunity to lay down new bone and make an abutment at each end of the fracture site. In dealing with areas of extensive mandibular loss, silver cap splints are made with a fine technique to completely crown the residual teeth of the mandible. These are cemented in place and thereafter give a firm means of attachment to all the types of bars and supports which may be required for fixation.

Injuries below the frontal bone and above the mandible are referred to in general as injuries of the middle third of the face. The bones comprising the middle third of the face include the maxillae, the nasal and malar bones, and the ethmoid bone. These bones have suture lines in common with the frontal bone. When injury causes fractures extending across this line, the case often becomes a neurosurgical problem also. Fracture of a single one of these bones often occurs in the simpler types of injury. The simple reposition of a single fractured bone of the middle third creates few problems. There are many acceptable techniques for each of them.

When the malar bone fractures, it usually does so at its articulations with the frontal, the maxilla, and the temporal bone and the crush is usually medialward into the maxillary sinus. Occasionally the bone itself is comminuted. For the simpler type of depressed malar fractures we have used the temporal method of reduction often known as the Gilles method. In it an

elevator is introduced beneath the temporal fascia and downward beneath the zygomatic arch. This allows firm leverage on the bone for elevation.

An occasionally badly comminuted fracture of the malar bone requires intra-sinus elevation. An approach is usually made through the canine fossa. Support is given to the comminuted fragments by a firm pack placed in the maxillary sinus after elevation. This method compounds the fracture, however, and damages the maxillary sinus mucosa, and the pack must be left in place for several weeks thus exciting a fairly marked reaction in the cheek. It should not be used in the reduction of the simple type of malar fracture.

We have used this intra-sinus method of elevation in some fractures of the malar bone seen as late as six to eight weeks after injury. Since it is the nature of the malar bone to heal very rapidly, malposition becomes firm after but a few weeks. Therefore, when a badly depressed malar fracture with loss of orbital support is seen after several weeks it is more than likely that simple means of elevation will not suffice. One is faced with the problem of accepting a deformity for later correction by transplant methods of fascia, cartilage, or bone or of re-fracturing the malar bone and elevating it into position. The latter method of correcting the deformity is preferable when it can be applied. After re-fracturing and repositioning, the bone must be fixed in its proper position for a long period of time. Fixation to a headcap by external means is best. An external pin couplet is a very satisfactory means of fixation and allows control of the malar bone for any type of manipulation to adjust its fineness of position postoperatively.

The nasal bones, due to their prominent position are, perhaps, the most commonly fractured bones of the body. The fractures range all the way from simple malposition correctable by elevation and thumb pressure to cases of extreme comminution and depression with disruption of the entire ridge support of the nose. The latter type is often associated with maxillary and ethmoid labyrinth fractures and with extensive lacerations of the soft tissues. In these latter cases the interior of the nose should be carefully repaired with replacement of the turbinates into their normal position and suture of the mucosa of both the septal and the lateral walls of the nasal space in an edge to edge manner. The nasal septum should always be replaced in a midline position. The Asch forceps is a useful instrument for gripping the septum and rocking it into place. The function of the nose and the appearance of the face should be equally important objectives in the reparative process. When the nasal interior is agglutinated into a mass of adhesions and scar tissue it can later be redeemed as an airway only through extensive sacrifices of intra-nasal tissue.

It is difficult in cases of severe comminution of the nasal bones to maintain the dorsum in its normal, ridgelike position during healing and some external means must be used to effect it. Perhaps the simplest method is the use of through and through silk or steel wire sutures placed at the superior and the inferior limits of the nasal bones and tied on both sides over a fashioned lead plate. This prevents broad spread of the comminuted nasal bone fragments and gives a tenting effect to the ridge. A more complex but more effective external appliance is required in some instances. It operates by means of two curved bars descending from a headcap and terminating in wire hooks inserted upward high into each nasal space. These are tensed and locked to give proper forward traction.

The ethmoid labyrinth when it is bulged laterally into the orbital spaces must be compressed medialward into its normal position by deep pressure at the inner angle of both orbital spaces. Simultaneous elevation of the nasomaxillary bone mass helps narrow the ethmoid. When x-ray reveals spicules of ethmoidal or frontal bone turned in an intracranial direction, or when there is evidence of a cerebrospinal leak, the services of a neurosurgeon are requested. The facial injury becomes a secondary matter. The manipulation of the facial bones should be deferred until the dural opening is well sealed. If it seems advisable, the soft tissues of the face may be repaired under local anesthesia in the interim.

When the maxillae are fractured, the problem of dental occlusion again arises. In those instances where the mandible is not also fractured one borrows the mandible to use as both a splint and a positioning agent by applying intermaxillary wires to occlude the teeth of the maxillae to the mandible. Because of the weight of this combined mass, one must then support the whole

lower face in order to keep it in normal relationship with the upper half of the face and head. There are many methods of attachment of the maxillary and mandibular mass to headcap supports to prevent sagging of the lower portion of the face. Here again the silver cap splint cemented to the teeth and attached to a plaster headcap with screw plates and bars lends itself to a satisfactory solution of the problem. Fine wires attached to the intermaxillary loops and run upward through the soft tissues of the cheek to a headcap bar can also be used to support the lower facial mass. The pull of such wires is entirely upward and if there is loss of bone substance in the middle third fracture a shortening of the vertical dimension of the face can result from this method. Vertical bar support which controls upward as well as downward movement is preferable in many cases. Rigid bars are applicable in this manner from a headcap appliance to the cap splints cemented to the teeth, or to an arch bar wired firmly to the teeth, or to external skeletal pin couplets inserted into the mandible.

Fractures of the bones of the middle third of the face tend in general to heal rapidly. Early and proper positioning therefore is imperative. Once healing in malposition has been effected it requires a major procedure to correct the deformity. Occlusion may be restored only by the sacrifice and grinding of teeth, and the use of cartilage or bone implant may be necessary to correct the contours of the face. Attention should be given to the facial injury as soon as the patient's general and neurologic condition permits. Improper healing in this area can result in gross malformations as well as in disabling malfunctions of the nasal and dental apparatus.

In the majority of fractures of the facial bones manual traction is adequate for replacement of the fragments. It is not uncommon, however, in fractures of the malar, the maxilla, or the mandible that application of average strength to the manual reduction is not adequate to restore the alignment of the parts. The use of steady traction is as useful in facial fractures as it is in fractures of bones of the other parts of the body. Elastic traction over hooks wired to the teeth and extending from upper teeth to lower teeth is frequently employed in reduction of mandibular and maxillary fractures. A severely impacted fracture of the malar bone can also be replaced by gradual rubber traction to a headcap appliance. In some very late impacted fractures of the malar or maxilla, however, elastic traction to a headcap is not adequate. There is a limit to the amount of pull which can be exerted from a headcap apparatus. When one has no success after twenty-four or forty-eight hours, other traction means must be resorted to. By means of perforating wires inserted into the infra-orbital rims or into the alveolar process of the maxilla or by means of couplet pins inserted into the body of the malar bone one can get a firm grip upon the structure which will withstand any ordinary pull over a long period of time. It is a simple matter then to construct a bed frame with pulleys and to attach a weight traction of several pounds to the wire loops or pin couplets. The head is braced into position with sand bags and one has all the necessary latitude in the direction and in the strength of pull. Most impacted fractures and those united in malposition beyond any other means of reduction will give with this kind of pull. Once the bone is restored to normal position it will either maintain itself there freely or it can be maintained by support to the headcap. The metal cap splint cemented firmly to the upper teeth lends itself admirably to this type of traction for disimpaction of the maxilla.

The soft tissue portion of the facial injury is often more readily repaired than is the injury to the skeletal parts. It is preferable to make the primary repair a meticulous and thorough procedure. To this end, it is best to cover the injury with sterile dressings until the patient is resuscitated and his airway established. He can then be transported to the place where his complete facial repair can be done.

The wound should be cleansed with saline irrigations and all the debris removed manually. In the face the soft tissue debridement can be done very conservatively because of the fine blood supply. Flaps and pedicled parts which in other portions of the body must inevitably be lost will often maintain their viability in the face. All completely free fragments of bone should be removed from the depts of the wound before the soft tissues are joined. Those fragments of bone which have even small attachements to the periosteum will usually remain viable and take part in the general skeletal healing if postoperative infection is avoided. Foreign bodies should be removed from the depths of the wound.

In uniting the soft tissues the buried suture should be held to the minimum requisite with maintaining the position of the flaps. Fine skin suture should be used for the skin edges and tension on these sutures should be avoided. They should usually be removed in three days. With careful hemostasis, drains are rarely needed and often are ill advised. In handling the soft tissue flaps the use of forceps should be avoided as much as possible. It is preferable to handle the flaps with the fingers or gauze. A great deal of tissue can be bruised in a long reparative procedure through the pinching of thumb forceps. When venous congestion and edema of the injured parts occurs postoperatively, the tissues tend to distort, healing is delayed, and a fine culture medium for postoperative infection is present. It is best combated by application of a pressure dressing to the postoperative wound. Healing is accelerated by the prevention of edema. When external fixation appliances are attached to the face, the application of a pressure bandage is extremely difficult, and it is not possible to wind an elastic bandage about the face. However, in circumstances such as this adequate pressure can be maintained by fashioning a portion of a rubber glove with vents and slits for the external appliances and with strings or straps extending posteriorly to tie behind the head. When large pedicled flaps of soft tissue have been formed by the wound the prevention of edema may make the difference between maintaining the viability of these flaps or losing the flaps through inadequate circulation.

The face is a sort of surgical paradise. It tends rarely to become infected. Infection can be a catastrophe, however. To prevent postoperative infection several points must be stressed. First, is the assured removal of all irritating foreign bodies whether they be completely separated fragments of bone, fragments of clothing, fragments of rock and earth, or of other material. The second important factor is the fixation of all movable fractured parts. A continued breaking down of fresh, new granulation tissue about fractured bone ends favors infection. The prevention of postoperative edema robs potential bacterial invaders of their favorite growth medium. When the wounds extend intra-orally as do so many of the facial wounds, mouth hygiene is of importance. All intraoral lacerations should be sutured with meticulous care. Mouth organisms can and usually do cause a very foul type of suppurating wound unless controlled. Normal saline is a most effective and non-irritating cleansing solution for use in the mouth. It should be used frequently throughout the day. The cleansing is followed with a light dusting of all intra-oral lacerations with a combination of penicillin and sulfanilamide powder. Severely wounded patients are given a routine post-injury medication of penicillin and oral sulfadiazine. In moderately severe wounds penicillin alone is often used. Penicillin solution is trickled into the lines of laceration after all reparative procedures. It is very desirable in all badly comminuted fractures that absolutely no infection supervene because the viability of a great number of small bone fragments depends upon just one nutrient vessel extending through its shred of periosteal attachment. Loss of this vessel means loss of the fragment of bone and in many extensive injuries loss of but a very few fragments of bone results in non-union.

Anesthetic choice is not easy in some types of face and jaw injuries. Some soft tissue work and some skeletal repair such as mandibular realignment can be done with local anesthesia or with morphine premedication. More extensive injuries require general anesthetic. This is particularly true when the wound extends into the airway and considerable bleeding is bound to occur during the surgical procedure. In such circumstances it is obvious that the airway must be occluded and intratracheal anesthesia either through the nasal or oral routes is preferable. When a tracheotomy tube is in place, most of the anesthetic problem is solved. We frequently use a combination of intravenous pentothal and inhalation nitrous oxide and oxygen. When working in or near the larynx, superficial application of a dilute cocaine solution combined with this anesthesia circumvents the laryngeal reflex.

The postoperative care of facial injuries is as important as the reparative procedure. It is important to maintain a clear airway and a dry lower respiratory tree. Since anesthesia need not be deep the patients awaken early after surgery and are encouraged immediately to cough and hyper-ventilate. The head is elevated as early as is possible to circumvent edema of the facial structures. This puts an added load upon the cilia in emptying the tracheobronchial tree, but

(Continued on Page 293)

THE Rh FACTOR

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Brief Review

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The Rh factor is, at the moment, one of the most discussed medical subjects appearing in the current medical literature. It is, perhaps, one of the most important discoveries of the present decade. Therefore, a brief review of the subject, and the presentation of a summary of the autopsied and recovered cases which came under my observation seem timely.

For many years there had existed in the minds of the investigators a growing suspicion, bordering on a conviction, that one of the major causes of fetal and maternal morbidity might, somehow, be linked with an incompatibility between the blood of the mother and that of the fetus, and a resulting antigen-antibody reaction. More recently, a few attempted to explain the pathogenesis of erythroblastosis fetalis on the same basis. Simultaneously, reports had appeared of the unexplained intragroup hemolytic reactions occurring in individuals who had received repeated transfusions, and in mothers who had given birth to erythroblastotic or macerated babies. In some of these cases, the presence of an agglutinin, which was thought to be responsible for the reaction, was actually demonstrated.

In 1940 came the announcement of the epochal discovery by Laudsteiner and Wiener of the Rh factor in blood, a Mendelian dominant, in about 85 per cent of human beings, which is capable of producing immunization in the remaining 15 per cent of individuals. Wiener was now able to explain the cause of the severe hemolytic reactions which occurred in patients who had received transfusions of homologous blood. Later in the same year, Levine and his co-workers published an observation that the sera of women who had had repeated miscarriages or abortions contained agglutinins shown to be related to the Rh factor. The following year, Levine, Katzin and Burnham published, in the *Journal of the American Medical Association*, an article entitled, "Iso-immunization in Pregnancy: Its Possible Bearing on the Etiology of Erythroblastosis Fetalis." This paper and several others which closely followed clearly established their theory that the genesis of erythroblastosis fetalis in a great majority of instances is related to the immunologic reaction due to the Rh factor between the mother and the baby. In spite of the importance of this discovery in clinical medicine, the medical profession in general was, at first, slow to grasp its significance. The clinical laboratory, too, was not immediately awakened to the importance of Rh determination in clinical medicine. The obstetricians and the pediatricians became more and more interested in the subject, only as they came in contact with clinical cases of their own, in which the Rh factor played a role. Only in recent months the interest of the physicians in general seems at last to have become more universal.

Today, thanks to the diligent and fundamental investigations of such men as Wiener, Levine, Diamond, Davidsohn and others, we have come to undertsand a great deal more about the Rh factor. At the same time, we have also come to realize that the subject has been, in a way, made so complex and complicated by the very same men who have been publishing new data as they come into their possession, sometimes too prematurely, that the rest of the medical profession has been left too bewildered to grasp the important theoretical and technical considerations which form the basis of the clear understanding of the subject.

The determination of the Rh factor and its related sub-types, the laboratory diagnosis of erythroblastosis fetalis and of the intragroup hemolytic reaction, therefore, constitute a complicated immunologic problem and their proper interpretation requires the understanding of genetics. The average clinical laboratory is neither equipped nor staffed to render complete immunologic service or genetic interpretation of the sub-

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ject which is yet undergoing further exploration by the fundamental investigators.

Fortunately, however, a few important practical observations along with the original basic discoveries can be and are being made which apply to everyday practice with satisfactory results and a degree of assurance. I shall discuss the laboratory phases of this complicated subject in a form of questions and answers, in so doing, purposely avoiding the use of genetic terms for the sake of simplicity.

1. What is the Rh factor? The name was coined by Wiener and Laudsteiner, the discoverers, and derived from the first two letters of rhesus monkey, whose blood was injected into rabbits to produce an anti-rhesus serum, which was found by them to cause agglutination of the blood of about 85 per cent of white individuals. The blood of these 85 per cent of people is known as Rh positive, because the anti-Rh serum causes its agglutination, while the blood of the remaining 15 per cent is known as Rh negative and is not agglutinated by the anti-Rh serum. The Rh positive is a Mendelian dominant while the Rh negative is a recessive.

2. What is the one most important basic question involved in this entire subject? The immunization of the Rh negative person by the blood of the Rh positive individual with resulting antigen-antibody reactions which may occur actively or passively, depending upon how the victim is sensitized.

3. What, then, makes the subject so complicated? Whereas, in the beginning, there was only one anti-Rh serum for the determination of the Rh factor, we now have three types of anti-Rh agglutinins, namely, anti-Rh$_o$, anti-Rh′, anti-Rh″, with occurrence of the last two, separately and in association with the first, giving rise to two additional anti-Rh serums, anti-Rh$_o$ + anti-Rh′ or anti-Rh$_o$′ and anti-Rh$_o$ + anti-Rh″ or anti-Rh$_o$″. Thus, we now have five major varieties of anti-Rh serums and their corresponding Rh factors instead of only one, and these five Rh factors give rise to eight types of human blood. Therefore, to make the complete Rh factor determination, we should have the three anti-Rh serums mentioned. However, they are not as yet available for routine use. The testing serum we now use contains anti-Rh$_o$ and anti-Rh′ agglutinins which gives about 87 per cent positive reactions on the bloods of the white individuals in New York City. Then we have the tedious procedure of titration for anti-Rh agglutinins, and the problem of dealing with the so-called "blocking antibodies" which interfere with the titration. We are also confronted with Hr factor, with whose nature we are not as yet quite familiar.

All of the procedures are complicated and exacting, requiring the attention of an expert technician, specially trained to do the job.

4. Briefly, what is the mechanism whereby the baby develops erythroblastosis fetalis? In 90 to 95 per cent of the cases, the father is Rh positive and the fetus is also Rh positive while the mother is Rh negative. The Rh positive blood of the fetus enters the Rh negative mother's circulation through the placenta and in time, causes in the mother the production of strong anti-Rh agglutinins. These agglutinins, in turn, enter the fetal circulation and cause a true hemolytic anemia through the destruction of the fetal blood and stimulate extra-medullary erythropoiesis. (See question 7.)

5. Are all Rh negative individuals easily sensitized by the Rh positive blood? No, only one in twenty-five to fifty are readily sensitized. Others may require several or many transfusions or pregnancies to become sensitized. The incidence of frank cases of erythroblastosis fetalis is, therefore, much smaller than generally anticipated, theoretically estimated to be one in from 200 to 350 pregnancies. Actual figures are still smaller. Levine estimated about 0.1 to 0.2 per cent of all pregnancies.

6. Can Rh negative mothers continue to have normal babies? The first child is expected to be normal provided the mother has had no transfusions with Rh positive blood before becoming pregnant. Several subsequent babies may be normal, depending partly upon how sensitive the mother happens to be and partly upon some yet unknown factors. Such mothers may have a normal baby again if the intervals between the pregnancies are long enough, that is, three to five years or longer, to let the anti-Rh agglutinins from previous pregnancies disappear. (This may be a theoretical wishful thinking, especially where the father happens to be homogygous Rh positive.) A normal Rh negative baby may be born of a sensitized mother, provided the father happens to be heterozygous Rh positive, in which case he has a 50 per cent chance of having normal Rh negative babies. The father may be considered

heterozygous if any of his blood relatives, namely, any one of the parents, brothers, sisters or children, should happen to be Rh negative. (It is also stated that the father is homozygous if his cells are agglutinated by Rh', Rh$_0$ anti-sera and by anti-Hr serum.)

7. May an Rh positive mother have an erythroblastotic baby? Yes, approximately 5 to 10 per cent of the mothers who have given birth to babies with erythroblastosis fetalis have been found to be Rh positive. This can be best explained, in most instances, as due to the presence of some of the sub-types of the Rh factor in the mother while other sub-types are present in the blood of the husband and in the blood of the fetus. In other instances the group A or B blood may act in the identical manner as the Rh factor in the baby's blood and cause the production of anti-A or anti-B agglutinins of sufficiently high titres in the group O mother to cause the disease. Some cases are also probably due to the Hr factor incompatibility. That is, in a few cases of Rh positive mothers of erythroblastotic babies, the agglutinins were found which reacted with all Rh negative persons and with some Rh positive persons. The Rh positive persons that reacted with this blood were heterozygous Rh positive, having an Rh negative recessive factor.

8. Can the presence of the anti-Rh agglutinins be demonstrated in every case? No, the titre may not be strong enough to be demonstrable. Occasionally, the "blocking antibodies" or the inhibitory substance in the serum to be tested may partially combine with the test Rh positive cells to neutralize the reaction. The technique has now been developed to eliminate this interference so that the titre of the anti-Rh agglutinins may be determined.

9. What are the "blocking antibodies"? They are the special Rh antibodies, incomplete antibodies, found in the sensitized maternal blood (along with the anti-Rh agglutinins) which combine with Rh positive cells, without causing agglutination, thus "blocking" or inhibiting the agglutination of the cells by the anti-Rh agglutinins. They are also known as the inhibitory substance.

10. Can the presence of the blocking antibodies be demonstrated? Yes, on the titration of the anti-Rh agglutinins, if agglutination of the testing cells occurs not in lower dilutions but only in higher dilutions, the presence of the blocking antibodies is suspected. Either the simple slide test of Diamond or the more complicated conglutination test of Wiener is employed to demonstrate them.

11. What is the Hr factor? It is difficult to define it because not enough is known about it. The symbol Hr represents the factor which is opposite to Rh because it is present in Rh negative blood. The Hr antibody clumps the blood from all Rh negative and heterozygous Rh positive persons. The failure of an Rh positive blood to be clumped by it indicates that it belongs to a homozygous Rh positive person. (This is still disputed by some authorities.)

12. Does the high titre of the anti-Rh agglutinins mean a bad sign for the unborn baby? Usually, yes, but not necessarily. The anti-Rh agglutinins may be carried over from previous pregnancies, apparently, with little effect on the unborn baby if it happens to be Rh negative.

13. Is early induction of labor or a cesarean section indicated in every case of mothers who have previously given birth to an erythroblastotic baby or babies or who are merely Rh negative? No. The Rh negative mothers who have had no history of miscarriage or of having been pregnant previously, should be left alone; only the development or rise in the anti-agglutinin titre should be watched. Even those mothers who have the history of having given birth to erythroblastotic babies or having had miscarriages or abortions, should better not be subjected to the risk of a cesarean section promiscuously. The insult of prematurity is usually a little bit too much for the babies already affected by erythroblastosis. If the titre of the anti-agglutinins should suddenly and sharply rise or fall toward the end of pregnancy, some investigators advocate a cesarean section. However, the low titre does not necessarily mean a mild form of erythroblastosis. A sudden drop in titre toward the end of pregnancy may mean hydrops of the severest form.

14. What would be the first step in treating a newborn erythroblastotic baby? The first step would be to do a blood count. If the count shows 3,500,000 of erythrocytes or higher, immediate transfusion is not necessary. Watch the count at frequent intervals. At the same time, have a Type O, Rh negative donor ready, if possible. Give Type O, Rh negative blood, approximately 10 c.c. per pound body weight, intravenously, and if urgently indicated, possibly through the umbilical vein. If an Rh negative donor is not available

TABLE I. ERYTHROBLASTOSIS FETALIS

Icterus Gravis

No.	42–1600	42–1657	43–98	43–2060	44–2180	45–78	45–381	Average
Term	prem	full	full	prem	full	prem	full	4f-3pm
Sex	F	M	F	M	M	F	M	4M-3F
Age	2 days	2 days	6 hrs.	13 hrs.	2 days	12 hrs.	3 days	34 hrs.
Weight	2,500	3,970	2,500	2,100	3,600	2,150	2,700	2,791
Rh	pos.	pos.	pos.	pos.			pos.	pos.
Liver	120 Gm.	220	205	100	110	132	129	145 (118.5)*
Spleen	24	43	22	21	30	28	18	26 (8.2)*
Ext. med. erythrop.	yes	yes	yes	yes	yes	yes	yes	yes

*Normal average weight

Data on Mothers

Age	29	29	35	35†	36	28	29	31
Grav.	III	II	II	III	V	V	IV	III
Children living	2	0	1	0	4	2	3	2
Dead	2	3	1	3	1	3	1	2
	1-2d-IG 1-mis-4m	1-sb-tox. 1-2d-IG i-sb-hyd	1-6h-IG	1-mis-6w. 1-sb-IG 1-13h-IG	1-2d-IG	1-sb-IG 1-sb-6m 1-12h-IG	1-1d-IG	
Rh	neg.	neg.	neg.	neg.	pos.	neg.	neg.	6 neg. 1 pos.

†Wassermann—positive

the mother's blood may be given. Such blood should be citrated, washed twice in saline and given resuspended, preferably in compatible plasma, or in concentrated form. In mild cases, one such transfusion is usually sufficient. The sooner the transfusion of Rh negative blood is given, the more effective is the treatment. Then, the mother's milk should not be fed the baby. The antibodies have been demonstrated in the mother's milk. The baby should be kept in an oxygen tent. If Rh negative donors are not available, compatible Rh positive blood may have to be given as an emergency measure. While Rh negative blood is said to survive for periods up to three months in the baby, Rh positive blood is known to be eliminated within four to five days. This is due to the demonstrated fact that the mother's anti-Rh agglutinins are present in the baby's circulation as long as twelve to fourteen days after birth. The essence of the treatment is, therefore, to keep up the oxygen supply to combat anoxemia. Plasma and glucose can be safely given as well as oxygen.

15. In what other conditions does the Rh factor play a vital role? In all cases of blood transfusion, the determination of the Rh factor has become as important as the routine typing and cross-matching of bloods of the recipient and the donors. This is imperative, especially when repeated transfusions are to be given to one recipient. Should he be Rh negative and the donors positive, precisely the same situation occurs as in pregnancy with the stimulation of anti-Rh agglutinins and a resulting hemolytic reaction in the recipient.

I wish now briefly to present a few pertinent data from the cases of erythroblastosis fetalis in which autopsy was performed.

Tables I and II give the summary of the findings in the thirteen cases of erythroblastosis, seven in the icterus gravis type, four of the hydrops type and two macerated babies. I wish to particularly call your attention to the last column of each group which gives the average figures. Your attention is directed to the following observations. Prematurity appears to be common, thus adding to the risk of the baby. The hydrops babies were born dead, or lived for one hour or less, while the icterus cases lived for an average duration of 34 hours; proper and adequate treatment might have saved some of these babies. The Rh factor was positive in all the babies tested. The liver was definitely enlarged, an average weight of 145 grams against the normal weight of 118.5 grams in the icterus series, and 191

TABLE II. ERYTHROBLASTOSIS FETALIS

	Hydrops					Macerated	
No.	37–441	40–2323	42–1178	44–992	Average	44–1519	45–1849
Term	full	prem.	full	prem.	2f-2pm	full	full
Sex	M	F	F	M	2M-2F	F	M
Age	1 hr.	sb	23 min.	sb	40m-2sb	sb	sb
Weight	4,100	1,800	2,500	2,950	2,855	3,500	3,250
Rh			pos.				
Liver	340	77	128	220	191 (129)*	165	112
Spleen	135†	12	10	40	49-21 (8.8)*	12	42
Ext. med. erythrop.	yes	yes	yes	yes	yes		

*Average normal weight
†This spleen was ruptured

Data on Mothers

Age	44	30	35	29	34	24	30
Grav.	III	VI	VIII	IX	VI	V	IV
Children living	0	5	3	4	3	3	1
Dead	3	1	6	5	3.7	2	2
	1-10d 1-sb-IG 1-1h-HY	1-sb-HY	1-sb-8m 1-12h-IG 1-4m-IG 1-4w-IG 1-23mi-HY 1-hyst-3m	4-sb-Mac 1-sb-HY		2-sb-Mac	2-mis 1-sb-Mac
Rh		neg.	neg.	neg.	neg.	neg.	neg.
Placenta	4 lbs.	3 times	2.5 lbs.				

grams against 129 grams in the hydrops group. The enlargement of the spleen is most striking in most of the cases. In the hydrops group an average weight of the spleen was 49 grams (including the one of extraordinary size, 135 grams, exclusive of which, the weight was 21 grams) against the normal of 8.8 grams. For the icterus group, the weight was 26 grams against the normal of 8.2 grams. Extramedullary erythropoiesis was observed in all or most of the visceral organs in varying degrees. Iron pigments were demonstrated in the liver, whenever stained for them. The brains were not examined for kernicterus.

Maternal histories revealed the following: The Rh factor on eleven mothers was negative, one positive, and one undetermined. Of the thirty-four dead babies of the thirteen mothers, five were hydrops (two stillborn), seven macerated (four by the same mother), three premature stillborn (no reference made as to erythroblastosis), one stillborn with toxemia, one unknown, one hysterotomy at fourth month, and four miscarriages. One of the women, thirty-five years of age, had four babies with icterus gravis and one hydrops and one stillbirth at the eighth month. Two of the five hydrops babies were delivered by a cesarean section. The mother of one of these two babies showed a low anti-Rh agglutinin titre and blocking antibodies, and a cesarean section was performed with a hope of saving the baby. The baby only gasped for breath and expired. One of the icterus babies had a syphilitic mother. The roentgen diagnosis of the baby's long bones was given as possibly syphilitic. The differential diagnosis was difficult. However, the microscopic findings of the visceral organs were those of erythroblastosis fetalis.

The first baby recorded here which came to autopsy in February, 1937, will be reported more fully. I believe this is the first case of erythroblastosis fetalis recorded in the Department of Pathology of the University of Minnesota. The baby was delivered of a multipara, aged forty-four, who had two previous pregnancies, one dying at the age of ten days, and the other a stillborn with icterus, and who had been given antisyphilitic treatment because of jaundice and death of the second baby, although her Wassermann was negative. The present baby showed a moderate degree of jaundice and a universal edema of the body with a marked distention of the abdomen which was first thought to be due to the distended

urinary bladder. When catherization failed, the abdomen was tapped, which revealed free blood in the cavity. At autopsy, the peritoneal cavity was filled with blood which had originated from a ruptured spleen weighting 135 grams. In this case, too, the question of congenital syphilis was at first entertained. Several of the mothers were suspected of having or actually had a hydramnios, and the placenta was swollen and heavy, weighing two to four times the normal weight.

Since 1939, we have recorded in this hospital approximately fifteen clinical cases of erythroblastosis fetalis of the icterus gravis type with complete recovery. Actually, I suspect that there must have been an equal number of undiagnosed cases, particularly among the stillborn who did not come to autopsy and whose mother's Rh was not determined. Several severe cases were encountered. Baby S. had 139,000 nucleated cells per cubic millimeter at birth (104,000 erythroblasts and 35,000 cells of the myeloid series) in June, 1939, and developed the most intense and sustained jaundice for more than five weeks. He developed a few petechiae, was lethagic and cyanotic. His hemoglobin dropped from 82 per cent to 30 per cent within a week. During the first eight days, four intramuscular injections of 28 to 35 c.c. of blood were given without appreciable improvement. On the ninth day, 100 c.c. of blood was transfused. The hemoglobin began to rise gradually up to 53 per cent on the twenty-fifth day. He was discharged on the thirty-second day, still somewhat jaundiced and anemic. Today, at the age of six, the baby appears normal and healthy. Baby G. was delivered by a cesarean section prematurely in November, 1940. It was sluggish and soon developed a deep jaundice. There were 48,000 nucleated cells per cubic millimeter. The hemoglobin was 48 per cent which gradually fell to 39 per cent, in spite of two intramuscular injections of 15 and 10 c.c. of blood and two injections of 20 c.c. plasma during the first week. A transfusion of 100 c.c. of citrated blood was then given, and the hemoglobin rose immediately to 80 per cent but slowly came down to 43 per cent on the twenty-sixth day. A second transfusion of 100 c.c. of citrated blood caused immediate rise of the hemoglobin to 89 per cent, where it remained until discharge. Samples of blood from the baby and the parents were sent to Dr. Wiener, who reported the mother to be Rh negative and the father and the child Rh positive. No diagnostic serum was available at the time and the Rh factor of the blood transfused was not determined.

I may briefly cite three illustrative cases in which a determination of the Rh factor was of utmost importance.

Mrs. N. gave birth to a hydrops and lost a considerable amount of blood. This was in the days when the importance of the Rh factor was not universally appreciated and no facilities were available for obtaining Rh negative donors. The clinician immediately ordered a transfusion which was done with the result the patient developed an anuria and elevated blood urea nitrogen.

In 1938, Miss X. was a patient at the age of eighteen with a diagnosis of encephalitis. She received four blood transfusions during her confinement in the hospital. She had since been married. Her first child died of erythroblastosis. She was found to be Rh negative.

Mrs. P. was admitted because of bleeding peptic ulcer. Her hemoglobin was 45 per cent. The usual blood groupings and cross-matchings were done, and the patient was receiving a transfusion of compatible blood. She developed a severe reaction before 100 c.c. of the blood had been administered. Regroupings and recross-matchings proved no error on the part of the laboratory. Then, Rh determination was ordered. This showed the answer. The patient was Rh negative.

In conclusion, I wish to recommend the following procedure:

Routine determination of the Rh factor in all hospital admissions may not be practical. However, in the light of our present knowledge and experience with the Rh factor and the intragroup transfusion reaction, it is recommended that the following procedure be followed, in order to prevent the transfusion reactions in which the Rh factor is found to play a decisive role.

1. All patients who come under the following category should have their Rh factor determined along with the usual blood grouping and cross matching, before the transfusion:

(a) All women who give a history of having been pregnant.

(b) All women who give a history of having had one or more miscarriages or abortions, or having had a baby or babies who had jaundice or died soon after birth.

(c) All patients who give a history of having reecived one or more transfusions, regardless of whether they had any reaction or not.

2. Every gynecological and obstetrical patient should have her Rh factor determined. If the factor be negative, it would be desirable to have her husband's and children's bloods tested for the Rh factor.

Clinical Discussion

E. M. Kasper, M.D.

Erythroblastosis fetalis is a term which personally I do not like. Hemolytic anemia of the newborn is preferable. My opinion is that hydrops fetalis is the most severe form, and then in decreasing grades come icterus gravis and congenital anemia of the newborn. I think they are all one and the same disease. Luckily we do not run into them very often, probably in only about 0.2 of one per cent of all births. This figure needs an explanation because, after all, 15 per cent of the mothers are Rh negative, and we wonder why we do not run into it more frequently. Individual variations in the permeability of the placenta may explain some of them. Also, there must be more than one pregnancy, as a rule, to produce an erythroblastosis. The modern tendency to restrict the number of children in the family is therefore a large factor in keeping down the number of erythroblastotic babies. Another explanation is that very often the condition escapes recognition. Some of these erythroblastotic babies are born with a mild anemia which, in the past, has not been attributed to the Rh factor. We also know that many husbands are heterozygous; that is, they have both the Rh positive and the Rh negative genes. In that event, some of the babies may be normal. It is interesting to calculate on the frequency of such marriages. Probably not over 5.5 per cent of all marriages are between men who are homozygous and women who are Rh negative, so the frequency would be cut down considerably just by that alone. Among American negroes and American-born Chinese, the incidence of the positive Rh factor is much higher. In the Chinese, the incidence of the Rh negative is said to be less than 1 per cent against 15 per cent among the white population. Many more male children are affected than female children. Another interesting point is in the recent work that some men are doing on feeble-mindedness. It was shown definitely from their study that there is some relationship between feeble-mindedness and this condition.

With hydrops fetalis, we have an obstetrical problem in which we often treat the mother for toxemia without really knowing the cause of it. There are a number of cases of hydramnios. The deliveries are very difficult, and the percentage of breech is increased, as are the other forms of abnormal presentation. Practically all babies die. With icterus gravis, on the other hand, we usually do not have very much of an obstetrical problem, because here the baby is not usually large and does not show any abnormality, except that it may be born jaundiced. The mortality among these babies was up around 75 per cent. It has dropped down now to about 15 per cent with the proper treatment. With congenital anemias of the newborn we do not have very much of an increase in obstetrical difficulty.

Regarding treatment, it is not clear just what we can do. We can say that the transfusions of Rh negative women with Rh negative blood should always be done. We have to be sure, however, that we do not use Rh negative donors who have been previously transfused with Rh positive blood, because they may transfer the anti-Rh agglutinins to the recipient. The transfusion of an erythroblastotic infant with Rh negative blood of the same group should also be done, if possible. Some men, on the other hand, claim that it is better to use Rh positive blood even though that blood is rapidly destroyed in the baby's circulation. The point raised by these men is that the Rh positive blood will neutralize some of the anti-Rh agglutinins in the fetal blood stream. Some men advocate using Rh negative and Rh positive blood alternately. We should never use the mother's blood, unless the cells are washed in saline before administration. The importance in treatment is to treat early and not to wait until the advanced symptoms are developed. The hospital should keep a list of Rh negative donors, and where we suspect the baby is going to be born

Dr. Kasper's paper has been somewhat shortened and the report of his cases is entirely omitted.

affected, we should have a donor ready. There has also been considerable discussion as to whether we should do cesarean sections on these mothers. I am not particularly sold on this idea, except in selected cases. The important thing in these cases is to watch the titer after the seventh month. Often we find that the titer does not rise at all; sometimes the titer will be rather high and all of a sudden it will drop; sometimes the titer will suddenly rise. There is no consistency. The only thing is to remember that when the titer does start rising, or if the titer does start falling, we have an indication of some trouble. When the titer falls, it means that the anti-Rh agglutinins are taken out of the mother's blood into the fetal blood, and these babies are apt to be hydrops.

The Rh tests are much more important during pregnancy than the Wassermann. I have found that, in over a thousand women, I did not find a single case of positive Wassermann where I had not suspected it before, or the mother knew it. With the Rh test, the percentage of the negative is much higher. I think the secret lies in trying to find some foreign material that is going to wipe out the anti-Rh agglutinins, but as yet we have not been able to find such a substance. It is important to tell Rh negative mothers with Rh positive husbands, that it is advisable not to have children too closely together, the consensus is about once every five years. That is in order to let the titer fall down in the mother's blood stream after pregnancy and to return to normal. The Rh negative mother should not nurse her Rh positive child. This condition is very interesting to the obstetrician, and as yet we do not have the answer. We hope that sometime or another we can show these mothers who are Rh negative and have Rh positive husbands that they can have a normal, living baby. The answer is not here today.

Differential Diagnoses—Kernicterus

Robert Rosenthal, M.D.

So much has been said about erythroblastosis that I think it is time we say something about the cases that are not. Therefore, I want to say something about the differential diagnosis. There are various conditions that look very much like erythroblastosis. First, common icterus neonatorum, simple physiological icterus, at times is very pronounced, particularly in the premature infant. It can be very pronounced and can appear very early, during the first day, and that is usually a characteristic of erythroblastosis. The premature infant, too, with icterus neonatorum may have more normoblasts than we usually find; i.e., over 5,000 per cu. mm. but if the number is considerably higher, the case should be viewed with suspicion. Usually we find also that when the number of erythroblasts have reached its maximum, it should return to normal without any particular treatment within a day or two, and there is usually no anemia in the first few days. Secondly, congenital lues can very easily simulate erythroblastosis fetalis. There may be icterus, splenomegoly, hepatomegaly, anemia, and a large number of circulating erythroblasts. Of course, the mother's Wassermann is extremely important because in the infant only a positive Wassermann can be depended upon. Dr. Ikeda mentioned something about the x-ray picture. That is very important because the x-ray pictures in erythroblastosis fetalis are just beginning to be studied and the changes are sometimes somewhat similar to those of congenital lues. Then, severe infections and particularly sepsis may produce a very severe icterus, enlarged liver, enlarged spleen, and often an increase of erythroblasts that appears several days after birth and usually appears after the onset of infection. Very often the focus of infection can be discovered. The septic temperature and the positive blood cultures help to clear up the diagnosis. Now, in the cases of congenital atresia of the bile duct, we have a very pronounced icterus, but usually there is no particular increase of the erythroblasts, though this can occasionally happen. The acholic stools are, of course, most important in differentiating this condition. Icterus usually appears a little later in the newborn period, but it may come at once. Then there is the large group of intracranial hemorrhage. That may at times simulate erythroblastosis fetalis, but the blood picture, the liver, and the spleen usually remain normal. Another condition, hemorrhagic disease of the newborn, may occur at about the same time as erythroblastosis fetalis. It may have a full-grown picture on the second or third day of

life, and hemorrhages may occur in erythroblastosis fetalis, though here the hemorrhages are usually of a terminal nature. It should be kept in mind, too, that erythroblastosis fetalis also shows a diminished prothrombin. Congenital heart disease can be confused with erythroblastosis. Edema may be present, and the liver and the spleen may be large. The erythroblasts may reach as much as 25,000 per cu. mm. on the first day. The differential diagnosis may be particularly difficult if there is an early physiological icterus. I think that during the past year, it has been shown that idiopathic cardiac enlargement is found fairly often in erythroblastosis; but in congenital heart disease, there is usually no family history, no hemolysis and no anemia. On the other hand, there is an increased hemoglobin and increased red blood count. Of course, the determination of the Rh factors of the parents of the patient are extremely important. I do not want to say any more about some types of congenital icterus that are very rare and are not of erythroblastotic origin.

I would like to comment on one of the symptoms of erythroblastosis fetalis, the so-called kernicterus. We have adopted the German name. We usually call it icterus of the basal ganglia. This condition was first described seventy-five years ago, but did not receive its name until 1903. It has been shown that there are certain portions of the brain, particularly the basal ganglia, the cornu ammonis, even certain parts of the spinal cord, occasionally become stained with bile. It is a very rare condition. In one very early series of 120 cases of severe icterus gravis only six cases show kernicterus. The interesting point is that kernicterus appears only in the newborn period, never in the adult. It never appears in an obstructive type of jaundice. It apparently appears only in cases of erythroblastosis fetalis. It is not a disease in itself. It is just one of the lesions found in these cases of severe jaundice. The cause has not been definitely determined but the one thing that we have learned is that apparently only abnormal cells, the cells that have become damaged, degenerated, or necrotic will be stained with the bile. Now, it seems that the damage of these cells has to take place before the staining takes place. The anoxemia comes in here. Children fairly often are born asphyxiated and these stained cells, these stained basal ganglia, are very sensitive to anoxemia. They are damaged very early. It was shown, too, about fifteen years ago, that these particular areas like the cornu ammonis have a particularly poor blood supply and therefore are particularly easily damaged by anoxemia. At present, the best hypothesis is that these ganglia are very susceptible to damage by anoxemia, and the staining with bile is secondary, but this hypothesis does not explain the almost 100 per cent association of kernicterus in erythroblastosis fetalis and its consistent absence in other conditions. Thus, a new hypothesis has come up claiming that apparently the anti-Rh agglutinins act with a specific toxin on those cells, the antigen sensitizes them, and then they become bile stained. The histologic picture is the demyelinization and degeneration of the nerve cells, and we designate its characters not on its pathologic picture but on the clinical picture. We may have early symptoms of lethargy, convulsions, twitchings, but very often the symptoms are not found until late. It may take weeks or months before the symptoms appear. The late symptoms are various kinds of apathy, choreiform movements, delayed static functions. Practically every case shows mental retardation. There may be varied degrees of spasticity. It is one of the conditions that we have not paid very much attention to, but we will have to consider it in any case of icterus gravis combined with erythroblastosis. There is one case on record, where a child died at the age of three years, a child who had had erythroblastosis and icterus gravis and was extremely icteric and showed the typical symptoms of kernicterus, but when he was autopsied, degeneration of the various areas was found, but no staining was found. These are to be found exclusively in cases of erythroblastosis.

General Discussion

DR. N. J. LILLEBERG: Originally, the Rh factor was of interest only to obstetricians and pediatricians, but now it becomes apparent that any Rh negative woman who is given blood either intravenously or intramuscularly is liable to become sensitized before her obstetrical careers begin. Levine has shown in a large series of cases that Rh negative women who had, in their first pregnancies, erythroblastotic babies, had been sensitized either in childhood or at birth. In one case, a mother received a transfusion fourteen years previously; her first baby was an erythroblastotic baby. But, for all practical purposes, we perhaps have to assume that once these mothers become iso-immunized to the Rh positive factor they remain iso-immunized the rest of their lives. As yet, it has to be proved that a number of years of lapse between babies will change that factor any. The antibody titer decreases after a few years or even after months, but the reticulo-endothelial system still retains the power to rapidly develop antibodies which again will

develop erythroblastotic babies. Levine has shown that 0.13 c.c., between 0.1 and 0.2 c.c., of blood will sensitize a mother and produce erythroblastotic babies. Therefore, if we are giving transfusions to Rh negative infants or children, we have to be very careful and give them Rh negative blood. In New York, during the plasma drive, somebody conceived the bright idea of suspending the red cells. Why throw away the red cells that we have been saving from the plasma? Suspend these red cells and give them to the charity hospitals and use them for transfusion purposes. Eighteen women who received these red cells aid were followed subsequently, gave birth to erythroblastotic babies in their first pregnancy. At the present time, we can only say that any mother who has one erythroblastotic baby should perhaps not become pregnant again.

DR. WOODARD COLBY: There apparently is still argument as to when to transfuse. When anemia is present at the time the infant comes under observation, then there is no question that prompt transfusion is advisable and this must be repeated at regular intervals until the blood destruction stops. Infants in a critical condition, or with bleeding tendencies, even in the absence of anemia, must be given small, frequent transfusions. Rh positive, Rh negative, and bank bloods have been used with good immediate results in raising hemoglobin and improving the child's clinical condition. But, the Rh negative blood transfusions caused a better sustained rise in hemoglobin, fewer reactions, and lower mortality. If the baby is not anemic, then the use of transfusion may be questioned. The hemoglobin and red counts must be done once or twice a day for about the first ten days and then as indicated. Vitamin K may be given to the blood donors and to the infant but the lack of a good response is probably due to a liver damage. In the Swendson case that Dr. Ikeda has on the board, that child was a very icteric child and the icterus and nucleated red cells lasted from three to four months. The mother told me today that for the first three years the child's teeth were very dark and bluish-black. Now, at the age of six, the teeth are nice and white and clean. I see that Dr. Ikeda's figures run almost equal—males to females. The general figure is about three males to one female, and in the fatal cases is about five or six males to one female.

DR. L. W. BARRY: Until we can desensitize these Rh negative mothers, until we can immunize them against the iso-agglutinins or anti-Rh bodies, we are up against it. I do not know how we can do it. I do not think cesarean section or watching the titer is any solution of the problem. When you do not know what else to do, you transfuse the woman. The patient gets transfused again and again and again. These women are going to be habitual aborters and are going to give birth to erythroblastotic fetuses. We may work on the problem and get something new, so that we can desensitize these women, but so far, I think that after a woman has had two babies she has had just about enough if she is Rh negative. I had one patient who had seven miscarriages, and I told her she could have a cesarean if she wanted it. She did not want it, and she delivered a macerated fetus. In another case, we were watching the titer, and we thought of doing a cesarean. She delivered a perfectly normal baby before anyone could get there.

DR. X: In a case where a woman had an Rh negative factor and had had a couple of dead babies, would you sterilize her?

DR. ROGER S. COUNTRYMAN: I did that. One of the dispensary patients had a history of five pregnancies without a living child; three were born living. She had nearly died of anuria with the fourth one following a transfusion, and we did not know about the Rh factor at that time. She was anuric for five days. When she became pregnant the fifth time, with the consent of the patient and her husband, I did a hysterotomy and sterilized her. I think it is justified in such a case.

DR. EDWARD SCHONS: In regard to the x-ray appearance, I think rather than a thickening of the cortex, what you have is a widening of the bone due to an increase in the medulla and a thinning of the cortex. You see that in the skull and in all the long bones. I do not believe that it simulates syphilis.

DR. W. RAY SHANNON: As regards decision whether Rh factor positive or Rh negative blood shall be insisted upon for transfusion of newborn infants showing erythroblastosis, I should like to raise a question. Presumably, an infant suffering from erythroblastosis, the moment his umbilical circulation is interrupted, is a little bundle of antibodies against Rh positive blood cells. Theoretically, at least, these must be neutralized before the infant can hope to carry on under his own power so far as red blood cells are concerned. They should be capable of neutralization by any Rh positive blood cells. It seems, on the surface, that the sooner that established mass of antibodies are neutralized the better for the infant. That would have to be fact unless the products of cell destruction became an important element as regards the safety of the infant. The only argument that those who would insist upon Rh negative blood transfusion can rely is the assurance that sudden release of the products of red cell destruction does result in greater damage to the infant economy than would a situation in which an artificial transport for oxygen were provided while a gradual destruction of the infant's own oxygen transport system went on. It has never been defined to me exactly where, in the process of red-cell development in the infant, the antibodies against his blood cells begin to take hold. If it should so happen that they act at any stage earlier than complete maturity then it should be evident that the quicker they were neutralized by such an antidote, say, as adult cells supplied by transfusion, the better. The answer to this question lies, in my mind, in the future.

Dr. Rosenthal mentioned kernicterus in connection with the form of jaundice we are discussing. He spoke of it as a postmortem finding calling attention to the fact the jaundiced condition of the brain cells did not occur except in brain cells previously damaged. I agree heartily with his implications that it is a postmortem diagnosis and not subject to clinical consideration.

DR. IKEDA: During the first few days of the baby's life, it is assumed that the degree of hemolysis of its blood is in proportion to the amount of the anti-Rh agglutinins in its circulation. The more anemic or jaundiced the baby, the higher the titer of the anti-Rh agglutinins in its blood. Under such a circumstance, it would seem of more practical and immediate importance for the baby to have a sufficient amount of Rh negative blood in circulation just to maintain its oxygen-carrying function to combat anoxemia, which is likely to be the direct cause of death. Rh positive blood, which is known to be destroyed in four or five days, is given to neutralize the anti-Rh agglutinins, but what about the supply of oxygen which is most needed by the baby? Furthermore, a sudden, massive destruction of incompatible blood within the baby's circulation may cause a shock which may have a disastrous effect on the baby. I would say that if at all possible, the first and immediate transfusion should be done with Rh negative blood. I am particularly glad that Dr. Rosenthal has given us a very comprehensive summary on kernicterus, which has been neglected by many of us who have become absorbed in the hematologic and immunologic aspects of the Rh fac-

(Continued on Page 284)

THE TREATMENT OF ACUTE ARTERIAL OCCLUSION OF THE EXTREMITIES WITH SPECIAL REFERENCE TO ANTICOAGULANT THERAPY

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ACUTE arterial occlusion of the femoral or brachial arteries by embolism or thrombosis is a serious medical emergency. Delayed, inadequate or wrong treatment for this condition often results in extensive gangrene.

In cases in which acute arterial occlusion develops a serious underlying cardiac or arterial disease usually is present and therefore the occlusion in the extremity may be only a forerunner of even more serious arterial occlusion elsewhere; for example, in the arteries of the brain, mesentery, lungs or heart. Many patients who have acute arterial occlusion are poor surgical risks because of the cardiac or vascular disease. If gangrene develops and amputation is necessary the patient may succumb in the immediate postoperative period.

Acute arterial occlusion of the extremities may be due to embolism or thrombosis. Arterial embolism occurs as the result of detachment of a thrombus in a fibrillating auricle (which is the most common cause), from a detachment of a mural thrombus which has developed at the site of a large recent myocardial infarct or from a detachment of a portion of a vegetation which has occurred in acute or subacute bacterial endocarditis. Two rare causes of arterial embolism are the detachment of a thrombus in a systemic vein and passage of the embolus from the right side of the heart to the left side through a patent foramen ovale, and the detachment of a thrombus in the aorta or a large artery which becomes impacted at a more distal and narrower point in the arterial tree.

Acute arterial thrombosis *in situ* develops most commonly as the result of arteriosclerosis obliterans at a point where extensive atheromatous change has occurred. It occurs less commonly as a result of thrombo-angiitis obliterans. In a third condition, simple arterial thrombosis, the thrombus develops with minimal or no apparent disease in the arterial wall. Simple arterial thrombosis may occur in blood dyscrasias, particularly polycythemia vera, as a complication of serious infectious diseases even in young adults, during congestive heart failure or as a postoperative complication. Rarely it occurs in young adults without known cause or antecedent disease.

The clinical manifestations of acute arterial occlusion are quite characteristic. Severe pain in the affected extremity usually is present and frequently, but not always, the pain develops quite suddenly. In a few cases the condition develops gradually in a few hours. Pain may be present in the entire leg or arm but usually it is most severe in the most distal portion. The extremity is pale and cold and the superficial veins are collapsed. Some degree of hypesthesia is usually present and anesthesia of the glove or stocking type may occur. There may be partial or complete loss of motor function. Below the point of occlusion arterial pulsations are absent. The diagnosis is based on these clinical manifestations.

Some important points in the physiology of acute arterial occlusion should be considered because they influence treatment. Ten years ago McKechnie and Allen[2] emphasized the fact that acute occlusion of an artery is almost always followed immediately by a severe arterial spasm which affects not only the main arterial trunks below the occlusion but also many of the collateral anastomosing arteries. This spasm is a major contributing factor to the severe acute ischemia of the limb. The spasm may endure for many hours before relaxation occurs spontaneously and, when the spasm finally does subside, severe damage may have occurred in the endothelium of the arteries distal to the occlusion as the result of prolonged ischemia. Secondary arterial thrombosis develops in these distal arteries when the spasm relaxes and, in such cases, extensive gangrene of the limb almost invariably follows. During the period of arterial spasm there may be considerable ischemia of nerve trunks which are very sensitive to deprivation of arterial blood supply so that, even though the circulation may

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ultimately be restored, ischemic neuritis will be a persistent residuum. Another factor which is important in treatment is that thrombosis may continue to develop at the site from which the embolus was detached and be a potential source for more emboli. In the case of occlusion by thrombosis *in situ* the thrombosis may progress in a proximal direction, occluding more and more of the collateral anastomosing arteries, and therefore may result in more serious and irreversible ischemia owing to organic arterial occlusion which is entirely separate from the secondary arterial spasm. For these reasons, two of the cardinal principles of treatment are: (1) relax the arterial spasm as soon as possible and (2) institute as rapidly as possible measures to prevent further thrombosis.

In their previous report, McKechnie and Allen also emphasized the seriousness of acute arterial occlusion by indicating the results in fifty-seven cases of arterial embolism and sixty cases of acute arterial thrombosis. The patients were treated in various ways, some of which, in view of more recent developments, were probably inadequate, and they did not receive anticoagulant therapy. In slightly more than 45 per cent of the cases of arterial embolism and in slightly more than 50 per cent of the cases of acute arterial thrombosis, gangrene developed. Of the forty-two deaths (36 per cent of 117 cases) in the period immediately following the acute occlusion, four occurred soon after the occlusion without gangrene; twelve occurred following amputation, and in twenty-six cases, gangrene was

TABLE I. ACUTE ARTERIAL EMBOLISM OF EXTREMITIES

Anticoagulant Therapy

Case	Probable source of embolus	Location of embolus	Duration of therapy, days	Result
1	Fibrillating auricle (rheumatic heart disease)	Right popliteal	14	Recovery of limb
2	Fibrillating auricle (rheumatic heart disease)	Right femoral	54	Recovery of limb
3	Fibrillating auricle (rheumatic heart disease)	Left femoral; both brachials	27	Recovery of all limbs
4	Fibrillating auricle (rheumatic heart disease)	Right brachial	10	Recovery of limb
5	Fibrillating auricle (rheumatic heart disease)	Left brachial	15	Recovery of limb
6	Mural intracardiac thrombosis (myocardial infarction)	Left femoral	11	Recovery of limb; death from cardiac failure

TABLE II. ACUTE ARTERIAL THROMBOSIS OF EXTREMITIES

Anticoagulant Therapy

Case	Arterial disease	Location of thrombus	Duration of therapy, days	Result
1	Thrombo-angiitis obliterans	Right femoral	37	Recovery of limb
2	Thrombo-angiitis obliterans	Right femoral	22	Recovery of limb
3	Arteriosclerosis obliterans	Left femoral	10	Recovery of limb
4	Arteriosclerosis obliterans; diabetes	Right femoral	14	Recovery of limb
5	Arteriosclerosis obliterans; diabetes	Right femoral	9	Gangrene; amputation
6	Thrombosis, postoperative; transurethral resection for carcinoma of prostate	Left brachial	7	Recovery of limb
7	Thrombosis, postoperative; exploration for carcinoma of pancreas	Left femoral	16	Recovery of limb; minor bleeding mouth and rectum
8	Thrombosis, postoperative; radical mastectomy for carcinoma of breast	Right femoral	7	Gangrene; amputation
9	Thrombosis, postoperative; exploration for carcinoma of pancreas	Left femoral	4	Gangrene; amputation; death 7 days later

present but death occurred before amputation of the affected extremity had been performed.

At the present time we feel that the following are important points in the medical treatment of acute arterial occlusion of the extremities:

1. Make the diagnosis early.
2. Start treatment immediately.
3. Do not elevate the extremity.
4. Do not apply heat locally in any form.
5. Use the Sanders oscillating bed in the maximal low foot and minimal low head position. If such a bed is not available, elevate the head of the bed twelve to fifteen inches.
6. Keep the room temperature between 80° to 85° F.
7. Give ½ grain (0.032 gm.) of papaverine hydrochloride intravenously.
8. Give 1½ fluid ounces (45 c.c.) of whisky every four hours.
9. Give heparin and dicumarol.

Attention is called to the specific point that the extremity should not be elevated. When acute venous occlusion develops, elevation of the extremity is advisable, but obviously elevation is never advisable in the presence of acute arterial occlusion because the markedly impaired blood flow through the arteries then will have to overcome gravity also and elevation may render a partially ischemic foot or hand more ischemic than before elevation.

Inasmuch as the extremity is cold after acute arterial occlusion, a rather widespread procedure among patients, and even physicians, is application of some sort of heat locally to the skin. This should never be done. The use of hot water bottles, electric pads, hot packs, bakers or heating lamps is absolutely contra-indicated, because after acute arterial occlusion the skin is very ischemic and burns will develop if a minimal amount of local heat is applied directly to the skin. Furthermore there may be hypesthesia or anesthesia of the skin and the patient may not realize that the skin is being burned. In many instances, gangrene has developed entirely as the result of such burns.

We feel that the use of the Sanders oscillating bed is superior to intermittent suction and pressure and intermittent venous occlusion, for the treatment of acute arterial occlusion of the lower extremity.

The high room temperature, papaverine and whisky are used to produce vasodilatation and are usually successful. However, if papaverine is not effective after the first dose, subsequent doses are rarely effective, and it is unnecessary to repeat the injection.

The plan for anticoagulant therapy is as follows[1]: Fifty milligrams of heparin should be given intravenously every four hours, 300 mg. of dicumarol should be given by mouth immediately and 200 mg. should be given on each succeeding day when the prothrombin is greater than 20 per cent of normal. This plan necessitates daily prothrombin time tests. The blood should be withdrawn for the prothrombin time test each day three hours after the intravenous injection of heparin. If, on the first day, the prothrombin time falls below 20 per cent of normal the administration of heparin may be discontinued. If this plan of medical treatment is instituted early, that is within a few hours after the acute occlusion has occurred, it is usually effective both in terminating the arterial spasm and in preventing further thrombosis. If the arterial spasm does not disappear within twelve hours after the treatment has been started, the sympathetic ganglia supplying the affected extremity should be anesthetized with a local anesthetic agent; and if this also fails to release the arterial spasm and the limb has shown no signs of improvement in the arterial circulation, embolectomy or thrombectomy should be seriously considered. However, embolectomy is rarely necessary and thrombectomy is not often successful.

We have treated acute arterial embolism in six cases and acute arterial thrombosis in nine cases by this plan of treatment. The results are summarized in Tables I and II. In these tables "recovery of the limb" means that gangrene did not develop, that pain was relieved and function was restored. It does not mean that the circulation to the limb was restored entirely to normal because usually there was persistent impairment or absence of pulsations in the affected extremity and some mild to moderate persistent arterial insufficiency. It will be noted that function of the limb was restored in all cases of embolism and in six of the nine cases of acute thrombosis. There was one death in each group. Bleeding as the result of anticoagulant therapy occurred in only one case and the bleeding was minimal.

(Continued on Page 280)

CLINICAL-PATHOLOGICAL CONFERENCE

DIAGNOSTIC CASE REPORT

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DR. A. H. WELLS: Case 3245 has proved to be one of our most interesting pathologic studies of the year. The clinical history has been summarized from the charts of three Duluth hospitals, yet there may be important missing symptoms.

DR. H. S. MARSHALL: This sixty-five-year-old retired storekeeper was admitted to the hospital November 19, 1945, two days before his death, complaining of severe nasal bleeding of two days' duration. He was quite uncooperative in regard to relating the nature of his illness. However, he did tell the nurse that he felt very weak from the loss of blood.

His past history included a ruptured, gangrenous, gallbladder with local abscess formation, in 1941. A cholecystectomy and drainage of an "orange"-sized abscess in the gall bladder area was performed at the Webber hospital. A duodenal fistula developed postoperatively, only to close spontaneously within one month. One year later a "grapefruit"-sized hernia protruding through the operative scar was repaired. In April of 1943 he sustained a fractured nose and a laceration over the right eye as the result of a fist fight. There was no loss of consciousness at the time. The fractured nose and laceration were repaired by his physician who later noted that—although the patient had been a rather obese, affable, personality with a tendency to heavy drinking—he became a distinctly disagreeable, good-for-nothing, chronic alcoholic who, in rapid succession, mortgaged his store and lost it and mortgaged his home and lost it, using the money so obtained to purchase, on the average, over one and a half quarts of whiskey a day. During this period of approximately two and a half years, he had had several attacks of what was considered intestinal obstruction, generally lasting two or three days. On three occasions he was admitted to a hospital where, following the use of Wangensteen's decompression tube therapy, he showed rapid improvement. The history relating to one of these admissions to St. Mary's Hospital reveals that he had had eight to nine emeses every day for six days preceding admission. There apparently had been no bowel movements during this period of vomiting. Headaches were not mentioned. He claimed to have refrained from using alcohol for three weeks. It was also learned that after his alcoholic bouts his vomiting and abdominal "gas" pains would become worse. He had had rheumatic fever twenty-five years previously.

From the Department of Pathology of St. Luke's Hospital, Duluth, Minnesota, Arthur H. Wells, Pathologist.

His physical examination was essentially negative. The blood pressure was 130/80 and temperature 100° F. The abdomen was soft, non-tender, and had no palpable masses. His white blood cell count was 10,400 with 75 per cent neutrophiles and 21 per cent lymphocytes. The red blood cell count was 4,300,000 and the hemoglobin 12.2 grams. The blood Kline test was negative. On the second hospital day he was greatly improved with gastric decompression therapy. His obstruction was relieved and he left the hospital on the third day.

On admission to this hospital, he was somewhat obese and his muscles were flabby. His white blood cell count was 13,350; red blood cell count 4,100,000; and hemoglobin 13.5 grams. There were 88 per cent neutrophiles. Urine examination was essentially normal. His temperature was 99° F., and respirations 18 per minute. The abdomen was relaxed and did not contain a palpable mass. There was marked pulmonary emphysema. Heart tones were distant and free from murmurs and irregularities in rhythm. The pulse was 80 per minute. The blood pressure on several occasions was 150/100. During his last two days there was no further nasal bleeding, and he rested quietly. There was no apparent suffering except for some nausea and undescribed pain in his abdomen. He vomited about 150 c.c. of light colored fluid about six hours before his death.

DR. A. H. WELLS: The case is now open for diagnoses.

PHYSICIANS: Coronary thrombosis with myocardial infarction; carcinomatosis of peritoneal surfaces; tuberculous peritonitis; high intestinal obstruction, due to adhesions; brain tumor; chronic subdural hematoma.

DR. A. H. WELLS: Dr. Tuohy's diagnosis is correct. Would you tell us what led you to the proper diagnosis?

DR. E. L. TUOHY: A serious change in character or mental status following an injury to the head, associated with recurrent evidences of increased intracranial pressure such as bouts of vomiting, should always make one consider subdural hematoma.

Necropsy

The necropsy revealed a remarkable combination of a cystic degeneration of the dura with a "subdural hygroma" on the left side and a chronic intradural hemorrhagic cyst, "chronic subdural hematoma," on the right side. The hemorrhagic cyst had caused a severe compression of the entire length of the right cerebral hem-

isphere so that its surface over a large area was concave instead of convex as seen in this kodachrome slide. The lesion extended from the anterior most frontal area to the posterior most occiput and involved about one-

Fig. 1.—Intradural hematoma with opened cyst wall exposing recent blood clots (x), margins (y) are separated to expose endosteal dura (z).

half of the distance between the sagittal sinus and the base of the skull along this entire distance. It measured from 1 to 2.5 cms. in thickness and was composed of a fairly thick-walled cyst demonstrated in this kodachrome picture (Fig. 1). The cyst was filled with a xanthochromic watery fluid with scattered small dark red blood clots (x). The latter totalled about 20 c.c. Many of these clots appeared to be of very recent origin. The thick cyst walls are incorporated in the split layers of the meningeal half of the dura. The meningeal layer of the dura has completely separated from the endosteal layer under the hemorrhagic cyst. (The meningeal and endosteal layers represent the two embryonic sources of the dura). Because of this separation of the two layers of the pachymeninx one may easily break through the meningeal layer of the dura at the cyst margin (y) and lift up the thick-walled cyst, leaving the endosteal layer (z) beneath, attached to the skull bone and having every gross appearance of normal intact dura.

Multiple histologic sections taken from the margins of the hemorrhagic cyst (Fig. 2) all reveal the location of the cyst within the meningeal layer of the dura (x). The endosteal layer (y) is completely uninvolved. Active fibroblastic organization of recurrent hemorrhages is most evident in this old cyst at its margin (z). It is in this new organizing connective tissue that one frequently finds huge congested and recently ruptured capillaries (Fig. 3), the obvious source of the recurrent hemorrhages within the old cyst wall.

In the superior frontal region of the left dura was a large flat cystic space filled with clear, watery fluid, measuring 6 cm. in diameter and about 5 mm. in depth. The fluid could be squeezed readily to other parts of the dura in which the layers appeared grossly intact. There was a mottling of brownish-yellow of old blood pigmeint and bright red of recent hemorrhages throughout the left frontoparietal region of the dura.

Many histologic sections taken from this entire area of the dura reveal an extensive cystic degenerative change primarily located between the inner third and the outer two-thirds (Fig. 4-x) of the meningeal layer (y) of the dura. The endosteal layer (z) has no significant degeneration. The meningeal membrane degeneration is evidenced first by fading and disappearance of fibroblasts, later by fading and disappearance of the collagenous fibrils and, finally, by the formation of cystic spaces (Fig. 5-x). These are at first microscopic in size and later grossly visible cysts. There is, in addition to this, the occurrence of small flare-like hemorrhages (y) at the margins of the normally present giant capillaries in the meningeal portion of the dura. Occasionally, a few red blood cells from these tiny hemorrhages have leaked into the cystic spaces. These giant capillaries and minute hemorrhages are seen in nearly every section taken from the frontoparietal regions of both sides of the dura. There are also many iron pigment laden phagocytes (z) —the remnants of previous hemorrhages. There is practically no inflammatory cell infiltration except for occasional lymphocytes in the immediate vicinity of the larger hemorrhages. The endosteal dura has no significant changes excepting for rare isolated pigment-laden phagocytes. A few pigment-laden phagocytes are also present in the arachnoid space in the sections from the cerebral cortex immediately underlying the hematoma. There are no significant histologic changes in the nerve cells, using routine hematoxylin and eosin stains.

Incidental necropsy findings included a grade II cirrhosis of the liver and mild toxic changes in the myocardium, liver, spleen, and kidneys. There were adhesions between the duodenum and bed of the gall bladder with some dilatation of the lumen of the duodenum but no evidence whatever of obstruction of any part of the small or large intestines. There were no diverticula, tu-

Fig. 2.—Margin of intradural hematoma showing endosteal dura (*y*) completely separated from meningeal dura (*x*). New granulation tissue organizing recent hemorrhages into cyst cavity at (*z*).

Fig. 3.—Giant capillary in organizing blood clot at cyst margin (Fig. 2-*z*) with surrounding hemorrhage and hemosiderin laden phagocyte.

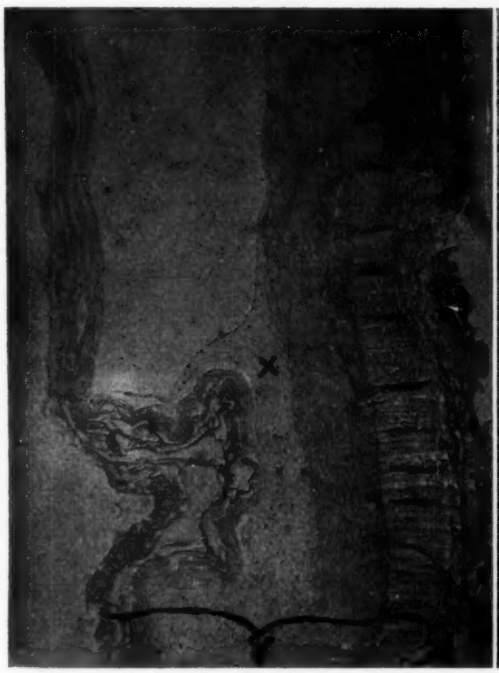

Fig. 4.—Cross section of pachymeninx revealing normal endosteal dura (*z*), meningeal dura (*y*), and idiopathic cystic degeneration of the meningeal dura (*x*).

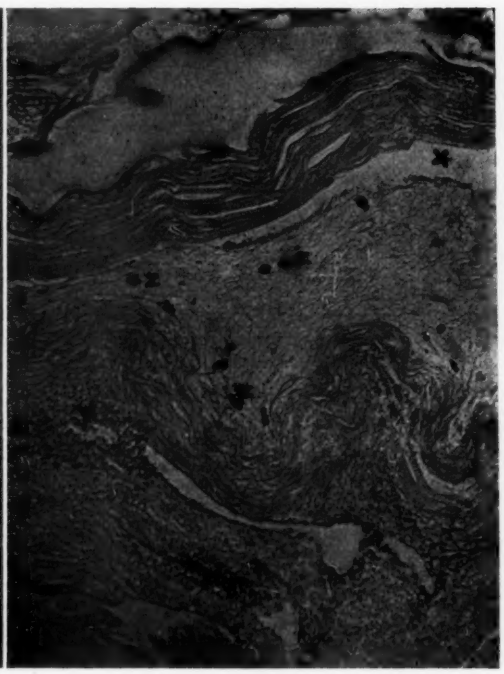

Fig. 5.—Meningeal dura with cystic degeneration (*x*), recent hemorrhages (*y*) from giant capillary and pigment-laden phagocytes (*z*).

mor masses, or areas of inflammation in any part of of the gastro-intestinal tract. There was no pneumonia.

Discussion

DR. D. W. WHEELER: Why wasn't the cirrhosis of the liver the cause of death?

DR. A. H. WELLS: The scarring of the liver was not advanced enough to be of great significance. In addition, there were only mild fatty changes in the liver cells. There was no ascites, edema, anemia, jaundice, gastro-intestinal hemorrhage, or dilatation of the portal tributaries.

DR. W. A. COVENTRY: How do you know that the hematoma caused death?

DR. A. H. WELL: The hemorrhagic cyst was quite large and had caused a great deal of compression of the right cerebral hemisphere. No other cause of death was found. The reason the patient died when he did and not a year previously may be because of recent hemorrhages into the cyst (20 c.c.) being incompatible with life, due to increased intercranial pressure.

Etiology

In spite of my limited experience with so-called chronic subdural hematoma and the present incompleteness of the literature survey of the subject, it is my opinion that the majority of neurosurgeons and pathologists have an erroneous conception of its histogenesis. At least, they rearely even consider the possibility of intradural origin.

The present most widely accepted concept of the origin of "chronic subdural hematoma"[28,33] includes: a subdural hemorrhage from injured subarachnoid veins, "bridging veins" or veins passing from the surface of the cortex to the saggital sinus; fibroblastic organization of the two surfaces of the blood clot from below into the dural surface and from its margins over the brain surface with capsule formation; central liquefaction of the blood clot and, finally, cyst formation.

In 1914 Korwitz[26] and later Hannah[16], Kaump and Love[21] and Baker[4] describe these hemorrhagic cysts as true intradural hematomas. In fact, Baker[5] feels that there is never a fibrous organization with cyst formation resulting from subdural hemorrhages, that the blood clots always absorb. To my knowledge, typical chronic hemorrhagic cysts have never been produced experimentally and are not found after craniotomy.

The many histologic sections taken from various areas of the dura of our present case and those of previous cases, reveal beyond any question, a sequence of events consisting of: (1) early idiopathic degenerative changes in the fibrous connective tissue of the meningeal layer of the dura; (2) cystic formations of varying size; (3) recurrent hemorrhages from the normal giant capillaries of the meningeal dura; (4) chronic hemorrhagic cyst formation within the meningeal dura with recurrent hemorrhages into the cyst. A somewhat similar process occurs in idiopathic medial necrosis of the aorta, resulting in dissecting aneurysm. When the facts are finally generally accepted, it is possible that all organized cystic collections of fluid and blood now generally considered to be subdural in origin are, in fact, intradural. In some cases, the basic disease process will be a cystic degenerative change of the meningeal portion of the dura, resulting in a splitting of this layer of the dura; in others there will be a splitting of the normal dura. The normally huge capillaries of the dura are subject to unusual forces not found in other capillaries of the body. These forces may be the result of changes in either the arterial or venous blood pressure of the brain or dura. They include stasis and engorgement of the capillaries due to back pressure in the veins of the dura and the frequently mentioned "dry cupping" or suction force exerted by changes in intracranial pressure. The actual hemorrhages from these capillaries may be precipitated "spontaneously" by these pressure sources or by a blow on the head, especially when the dural capillaries are engorged. Intradural hemorrhages of the newborn and of younger adults will be the result of the giant capillary ruptures with hemorrhages splitting the meningeal layer of the dura or a separation of the two embryonic layers without a preceding cystic degenerative change in the dura. These layers at times can be split normally without much difficulty. True subdural hemorrhages which do not kill will be eventually absorbed without cyst formation. Hemorrhagic diseases of the blood such as purpura hemorrhagica, scurvy, and leukemia, predispose to intradural hemorrhage. The habitual use of alcohol not only predisposes to head trauma but also either accentuates idiopathic cystic degeneration of the dura, or in some way enhances intradural capillary hemorrhages. Infectious processes of the dura of known and unknown etiology may rarely be important factors in intradural hemorrhage.

Pathologic Anatomy

Hemorrhages, in relationship to the meninges, must be classified as: (1) epidural, (2) intradural, (3) subdural, (4) subarachnoid or intrapia-arachnoid, and (5) subpial. We are concerned here with those cases in which a chronic hemorrhagic cyst formation attached to the dura is the ultimate result of the hemorrhage. These hemorrhages are nearly always located in the fronto-parietal areas over the convex portion of the cerebrum. Rarely, they are found in the occipital, subtentorial, basal, and spinal areas.[2] In a review of 204 cases described by ten authors, sixty-one, or one-third, were bilateral.[4] The gross appearance depends to some extent upon the age of the hemorrhage. Those of very recent origin will have a fresh dark red blood clot and, even if explored on the day of the injury, or within two weeks of the time of the injury, there will always be a limiting membrane holding the blood clot in the fronto-parietal area and preventing the action of gravity. Munro and Merrit[28,33] describe an inner limiting membrane of fibrin, which is later organized by fibroblasts. This is also repeatedly theorized and described by other authors as the origin of the inner membrane and, of course, touches the very heart of this controversy. There is central liquefaction of the blood clot and fibroblastic organization of the peripheral parts of the clot. New giant capillaries are formed. Finally, the wall becomes a thick, poorly cellular, hyalinized layer or

laminated layers of fibroblastic tissue, which may even become calcified. Recurrent hemorrhages into the cyst cavity are common, so that the contents of the cyst may vary from a slightly xanthochromic fluid to a solid mass of blood clot in various stages of decomposition. A detailed study of the histopathology has been described in connection with our present-case study.

Clinical Manifestations

Although in any one physician's practice, cystic hematomas of the dura are rare, their potentialities are such that a thorough understanding of their clinical recognition is mandatory. In one analysis[22] of 245 cases of various authors 82 per cent were in adults and of these 87 per cent were males. There is a peak of frequency during the first year of life, and the greatest incidence occurs between the fourth and sixth decades. The history of mild trauma[5,6,7,35] preceding the onset of symptoms and signs is a generally recognized feature. Only a small percentage of the patients suffer from fractures of the skull.[6] In some patients there may be no signs or symptoms of the encapsulated hematoma.[24] However, in the majority, a thorough study of the case should lead to suspicion of the lesion, necessitating bilateral trephinement for the diagnosis. Any discussion of the subject must be divided into the manifestations evidenced in adults and infants separately. The clinical findings in adults are mainly dependent upon a rapid to extremely slow increase in intracranial pressure brought to bear primarily in the fronto-parietal regions. They are very protean and characteristically fluctuate from time to time. The condition should always be considered in patients with cranial trauma in which the symptoms do not subside after months, or even years, following the injury. In a careful study of forty-two cases of "chronic subdural hematoma" Jelsma[20] found the following frequency of clinical phenomena: mental changes 86 per cent, headaches 79 per cent, drowsiness 79 per cent, motor disturbances 70 per cent, latent period 70 per cent, coma 57 per cent, reflex changes 56 per cent, cranial nerve disturbances 47 per cent, and a lucid interval 45 per cent. Of the less frequent manifestations having a mean average of 19.7 per cent, he included in order of their frequency: choked disc, vomiting, pulse changes, increased temperature, sensory disturbances, nystagmus, retinal hemorrhage and fractured skull (4.9 per cent). Among the more frequent mental changes are failing memory, confusion, full blown psychoses, uncontrollable irrationalism, excitement, serious character depreciation, and changes in spontaneity of personality.[1,15,17] The entire gamut of psychiatric abnormalities are possible, including neurosis, mania, depression, paranoid delusions, and gross deterioration.[9] Among 3,100 consecutive autopsies of psychotic patients there were 245, or 7.9 per cent subdural hematomas.[2] Effects referable to the cranial nerves frequently include diplopia, failing vision, hemianopsia, tinnitus, and decreased hearing. A homolateral dilatation of the pupil was correct in localizing the hematoma in over 50 per cent of the cases.[1,3] This sign is explained[15] by the fact that the brain stem is shifted toward the opposite side of the tentorial opening. Later the hippocampal gyrus is forced between the brain stem and the margin of the tentorium on the same side, pressing on the third cranial nerve, causing dilatation of the pupil. The frequent finding of homolateral nerve disturbances, especially that of hemiparesis, is described as being due to either pressure of the margin of the tentorium against the brain stem on the opposite side of the lesion or else pressure of the motor cortex against the skull on the opposite side. In sixty-two cases[31] 11 per cent had visual field defects. In a review of 244 cases from the literature, 44 per cent were described as having choked discs.[23] Practically all authors agreed that in adults the diagnosis can be most easily established by bilateral trephine openings. However, many other adjuncts have been used to advantage. Encephalographic studies are recommended by some[27] and discouraged by others.[38] Generally, they are not found very useful. Psychometric tests were found of considerable value in relationship to war injuries.[1] Dural hematomas result in syndromes which can be psychiatrically distinguished from war neuroses.[1] The electro-encephalogram produces a wave pattern in cases of subdural hemorrhage which is not pathognomonic of the condition, but a valuable adjunct in its diagnosis.[36] An x-ray demonstration of the shift of the pineal gland[10] has some diagnostic value. The condition must be differentiated from brain tumor, cerebral arteriosclerosis, or thrombosis, postencephalitic syndromes, and persistent headaches of unknown origin.[18]

Because of the frequency of subdural hematomas in infants, the great likelihood of the lesion's interfering with a rapidly growing brain, the bizarre clinical manifestations, and the ease and safety with which the lesion can be diagnosed with certainty by the use of bilateral needle puncture between cranial sutures into the dural cyst, as emphasized by Ingraham and Matson[18], it is absolutely essential for physicians caring for infants to have a thorough understanding of the condition. The generalized symptoms, such as fever, vomiting, hyperirritability, and failure to gain weight, are frequently found alone or in addition to the more specific neurologic findings of convulsions, stupor, and paralysis. All hydrocephalics should be considered possible cases of subdural hematoma since there frequently results a separation of the cranial sutures and bulging of the fontanelles, as the result of dural hemorrhages.

Treatment

Once the diagnosis is made, all authorities agree that operation is indicated. The points of disagreement concern the size of opening through the skull, how much of the cyst membrane must be removed, and whether drains should be left in place or not. Several outstanding neuro-surgeons[20,24,25,35,39] feel that an osteoplastic flap with more or less complete removal of the hematoma and its walls is advisable. Many others[8,10,22,37] feel, for the most part, that the more simple procedure of trephine openings with irrigation and suction of the contents of the cyst and the removal of lessor amounts of the cyst wall is adequate in the majority of cases. All authorities agree that bilateral bur openings are indicated in the fronto-parietal area. The treatment of chronic dural hematoma is different for patients with fused cranial bones, as compared to that most efficacious for infants. With an experience of ninety-eight cases

of subdural hematoma in infants, Ingraham and Matson[19] have advised a gradual decompression of the dural hematoma by the repeated aspiration of the contents of the cyst for a period of a week or two preceding craniotomy and complete removal of the cyst wall and contents. The gradual decompression is easily carried out with a syringe and needle and allows a period of improvement in the patient's general condition before the more extensive surgical procedure.

Summary

The case of a sixty-five-year-old chronic alcoholic with a chronic intradural hemorrhagic cyst, the immediate cause of death, on one side and a chronic intradural cyst on the other, has been described. There was an injury which may have precipitated the hemorrhage two and a half years before his death, at which time he had a serious change in personality and began experiencing repeated attacks of vomiting.

The histogenesis of the chronic hemorrhagic cyst of the dura in this case was traced to an extensive cystic degenerative disease of the dura. The question is raised whether so-called chronic subdural hematoma ever arises from true subdural hemorrhages.

A brief review of the etiology, pathologic anatomy, clinical manifestations, and treatment is included.

Necropsy Diagnosis

1. Idiopathic cystic degeneration of the dura.
2. Chronic intradural hematoma (immediate cause of death).
3. Compression of right cerebral hemisphere.
4. Intradural hygroma.
5. Cirrhosis of the liver.
6. Chronic alcoholism (from history).

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FACTORS IN RURAL HEALTH IN MINNESOTA

(Continued from Page 226)

tracts at a cost which is not excessive, will be available to all of the people of Minnesota. It is anticipated that under these contracts, free choice of physician is assured. Also, it is at present anticipated that the contracts will be available to virtually any citizen of the state. Until all of the details are finally determined, it is not possible to predict the costs nor the coverage under the contract. It is expected that the plan will be a distinct step in the right direction of providing adequate, good medical care at low cost.

In conclusion, then, it seems apparent, first, that good medical care is available to all people of the state; and, secondly in the not-too-distant future, adequate, good medical care will be purchasable at a cost which any or all of the people of Minnesota can afford to pay.

NOTES ON THE HISTORY OF MEDICINE IN HOUSTON COUNTY PRIOR TO 1900

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(Continued from February issue)

Franklin H. Whitney, born probably about 1862, was a graduate of the Hahnemann Medical College of Chicago in 1883, and it is recorded that on May 27, 1885, he received state license No. 1061 (H) to practice medicine in Minnesota. Shortly afterward he settled in La Crescent, Houston County, a village whose early hope of metropolitan growth had not materialized, and he remained there an undetermined number of years. In 1895 he served as county coroner. Information has not been available as to his career thereafter.

J. R. Wilson, a practitioner of the regular school of medicine, was certified to practice medicine in Minnesota under the Affidavit Ruling of 1887. There is record that he was in Pickwick, Winona County, in 1890, and earlier, and that in the middle nineties he was in Hokah, Houston County.

* * *

In the conduct of research in relation to early physicians in several counties of southern Minnesota it has become evident that there were many practitioners, unchronicled in official directories and reports, who drifted across the region, staying a few months or a year, leaving mention of their names in local newspapers. Some of these men were ignorant opportunists, some were sincere but incompetent, others were well trained in their day. From this mingled group some few names have appeared in these notes on physicians of Houston County, and it may well be, as was suggested earlier, that there were others of whom record has not been obtained and others still of whom no record exists. Of the able and stable practitioners, however, who maintained and advanced their profession in Houston County, it is hoped that the roster is complete.

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NOTES ON THE HISTORY OF MEDICINE IN FILLMORE COUNTY PRIOR TO 1900*

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"..... I have been able to derive much information, which if not always exact, was indicative of truth."

WILLIAM W. FOLWELL,
A History of Minnesota, 1926, Vol. 1, page xii

IN 1850 the region that soon was to become Fillmore County, Minnesota Territory, had lain for centuries in its natural beauty and productiveness, almost untouched save by time and weather. Its only inhabitants had been the wild animals that made it their home and the nomadic Indians, scarcely less wild, who roamed over it on foot or on their half-tamed ponies. In the opening decades of the nineteenth century there had crossed it, perhaps occasional white explorers and trappers.

As the fifties began to unroll, white men coming into southern Minnesota in search of homes found this region, both lovely and stern, to their liking. Of rich soil, usable limestone, abundant streams and varied woodland, it offered all they sought. Nor were they blind to the freedom of its upland prairies and the rare scenic beauty of its forested slopes and its river valleys, precipitous in the eastern portion as they neared the Mississippi River, shallower in the west; nor to the curious interest and the possible usefulness of those geologic phenomena, the "sink holes," caves and underground passages of the western stretches. Not much later the county was to be recognized as a paradise for geologists because of its ancient and varied formations and no less as a paradise for botanists because of its lush production of grasses, flowers, vines, nuts and fruits, and of trees valuable for timber, growth that even to the untutored eye was fair promise of the friendliness and bounty of the land when cultivated crops and orchards should have been introduced.

Beginning in 1851 the settlers came into what is now Fillmore County, as into all southeastern Minnesota, at first slowly, then in constantly increasing numbers, entering by the route so often mentioned: Overland to points on the eastern shore of the Mississippi River, in Illinois and Wisconsin and as far south as Kentucky and Tennessee, and thence by boat to points on the western bank.

*In a preceding article on the history of medicine in Houston County there was included much material relating to general history and medical history common to Houston and Fillmore Counties and other counties of southeastern Minnesota that will not be repeated here. In the present article appears new material which in turn applies not to Fillmore County alone but to Houston County and other counties of the region as well.

†A member of the editorial department, 1916-1919; 1919-1939, personal secretary and assistant to the late Dr. William James Mayo.

Many landed in Iowa, many in Houston County, Minnesota, and others came down from the little settlements of St. Paul and St. Anthony, which they had reached by northern routes. All went first to the land office at Brownsville, Houston County (later the office was moved to Chatfield, Fillmore and Olmsted Counties), and from there fanned out into the waiting land. One immigrant of these hundreds, and he must have been typical, traveled by railroad "from a town in Wisconsin to the Mississippi (LaCrosse), then took steamboat to Brownsville, then the stage to Elliota (in Fillmore County) and then on foot over the prairie" to his destination farther west.

Fillmore County, which was named for President Millard Fillmore, is the second county of the state from the Mississippi River in the tier that borders Iowa. On the east is Houston County, on the north are Winona and Olmsted Counties and on the west is Mower County. The boundaries of Fillmore County, approximately as they exist today, were established by an act of the state legislature on February 23, 1854, whereby they were much restricted from a widely comprehensive survey of a year earlier. To touch briefly on the controversies that the county experienced over the location of the county seat. First established in Winona, early in 1853, the county seat was transferred to Chatfield, obviously an illogical location, on the northern edge of the county, in December of that year; from March 2, 1855, to April 25, 1856, it was in Carimona, almost in the center of the county; on April 26, when public vote had fallen for Preston, about four miles northeast and more nearly central, Mr. John Kaercher, a substantial citizen of the county, later the founder of Clear Grit, but in 1856 in Preston, sent a man and team to Carimona for the records, which were conveyed to Preston in two coffee sacks.

The settlers entered the borders of Fillmore County on foot, with oxen, horses and in a few instances with few mules. These pioneers were of many nationalities, English, Scotch, Welsh, Irish, Bohemian, German, Dutch, Danish, Swedish and Norwegian, and whole clans as well as individual adventurers were in the movement from east to west. Many came directly to Minnesota from their native lands across the Atlantic; others were from families who had lived for a generation, or several generations, in New England or along the Atlantic seaboard; still others had pioneered in the thirties and forties from the East into Ohio, Indiana, Wisconsin, Michigan and elsewhere. Fillmore County became, and has remained, one of the most populous counties in the state; there were 13,542 inhabitants in 1860; 4,000 more in 1865; and in 1875 there were more than 28,000. From census to census since 1870 the population, preponderantly Scandinavian, has not varied more than 4,000; the peak was reached in 1895 with a population of 28,599.

Although the settlers retained many of the Indian names long established in the territory, they stamped on their new land their own names and nationalities, their hopes and homesickness, their imagination, political beliefs and religious faiths in the titles that they gave to natural formations and to civic divisions and organizations, as one may see who scans a map on which creeks, rivers, swamps, rocks and hills, townships and villages are indicated.

These people were of all occupations and professions. They promptly cleared and planted land; established local governing boards; built homes, many schools, and churches of a dozen denominations. Those from New England, especially, evincing hope and faith in the local water power, founded mills of different kinds, utilizing every stream; disappointingly soon the water power proved to be inadequate and all but a few mills and waterwheels eventually were abandoned. In some communities vestiges of the dams and wheels can still be seen. They

opened manufactories of the staple articles needed by all: woolen material, furniture, brooms, barrels, boxes and shingles, wagons and sleighs. They opened stores and blacksmith shops and tin shops. They helped to promote and support a network of stage-lines that carried mail and throngs of passengers.

In Fillmore County, as in contiguous counties, to meet the needs of the many travelers there came into being wayside taverns which provided stables and "trusty ostlers" for the horses and extended almost fabulous hospitality to the public. To name only a few of these inns, famous throughout the state, which still live in memories, there were the Carimona House, in the village of Carimona, and the Stanwix in Preston, the Medary House in Chatfield. The famous Old Territorial Road was surveyed in 1855 over a trail of earlier use and passed from St. Paul through Cannon Falls, Oronoco and Rochester and, in Fillmore County, through Chatfield and southeasterly across the county into Canton Township and on to Dubuque, Iowa. Another route in the county followed the South Branch and the main stream of the Root River, passing the sites of Rushford, Carimona and Forestville, and gave access to La Crosse through Hokah on the east and to Mankato through Blue Earth on the west. In the southern part of the county and the northern part of Winnesheik and Howard Counties, Iowa, the pioneers reached Brownsville, Houston County, and La Crosse over the Norwegian Ridge Route. One branch of this road, in southeastern Fillmore County, passed through Elliota, south of the present town of Canton (founded in 1879), and, once in Houston County, turned northeast. The other branch, a few miles north, originated in the south central part of Fillmore County, passed through Lenora and Newburg and entered Houston County near Rushford. These two branches converged on Diamond Ridge and separated at Caledonia, diverting traffic respectively to Brownsville and to Hokah and La Crosse.

In Fillmore County physicians and lawyers among the settlers opened offices and hardy editors founded newspapers, which sheets were of varying fortunes. One proprietor said plaintively, "As is well known, there is a great mortality among new newspapers, the death rate exceeding even that of the human race; in fact, as many newspapers relatively die before five months of age as children before five years." But at least they disseminated information to the eager pioneers. And some of the utterances, often rhetorical, of editors give now much knowledge of conditions then, as for instance, about transportation. Among the stage-lines and mail routes through the county were those of O. M. Walker, a well-known citizen in Minnesota and Iowa. Our plaintive editor expressed himself thus to Mr. Walker: "Why is it that it requires from seven to ten days for a newspaper or letter to find its way to Chatfield, through the mail, from St. Paul? We do not like to be controversial, finding fault, but it certainly demands inquiry, when it only requires from 24 to 30 hours to make the trip between these points, by way of Winona, the present route of the mail. Again, how does it happen that the Chatfield mail is sometimes carried through to Rochester before we receive it? Mr. Walker will please take notice." Mr. Walker, apparently, did not take notice, since more of the same appeared.

Old files of newspapers from the fifties through the next few decades give the reader an absorbing and vivid picture of the life of the times; give endless material that could be woven into the fabric of an anecdotal history. Those were the years when county superintendants of schools stressed in their printed reports to the public the value of reading, writing and spelling and spoke scathingly of teachers who were delinquent in imparting knowledge of these subjects; when a ten dollar bill was a "Sweet William;" when photographers were "daguerrean artists;" when farmers advertised for lost martingales, for strayed

horses and, particularly, for lost oxen. For instance, "one red ox, with staggy horns, white spot on face and white spots on the belly and hips, a long red tail with a white end," happily was recovered, a year after it had strayed, in a township in another part of the county. And a team of working oxen was lost; the near ox, light brindle with a large white spot on the forehead, the off ox, dun color with a small white spot on the forehead; both oxen had brass knobs on their horns and their yoke was a new red one with a patented bow key. Every druggist announced that strychnine was always on hand, presumably for the extermination of animal pests on farms; or perhaps, it has been suggested, because it was used in medical practice then more than now and was hard to get. When a druggist, particularly a physician-druggist, had a good supply he made the fact known. There were fascinating advertisements of Yankee notions, magazines, clairvoyants and hair restorers; patent medicines, painkillers and advice to opium eaters, cancer cures, even as now, and casual mention, not as now, of the outbreak of diphtheria or smallpox in a vicinity. By the early eighties there appeared an occasional advertisement by a physician for a lost instrument: a hydromatic syringe, a perforator used in cases of embryusterulcia, an aspirator used in cases of hydrenleroma phalocele, a myopodiartotican used in cases of myopia and a dentiscalpium used in cases of calcareous precipitation. (The spelling and definitions are as they were printed).

With the coming of the Civil War, when Fillmore County was eight years old and not yet recovered from the panic of 1857, there was much newspaper comment, such as (in 1862), "Ho! for the War! Intervention of England and France. 50,000 more soldiers. Call for a sixth regiment of Minnesota Volunteers;" about enrollment boards, exemptions from service, enlistment of troops and paying of bounties to stimulate recruiting. Although an Olmsted County newspaper commented disparagingly on men from Fillmore County who presented themselves before the Enrollment Board of the district, in Rochester, for exemption, Fillmore County nevertheless was represented with honor in the Union Army in many regiments. Company A of the Second Minnesota Infantry, for example, was made up chiefly from Chatfield; and although the famous Rangers of the county were not in the war, they were on duty within the state. Immediately after the war politics waxed and waned; during the presidential campaign numerous Grant and Colfax Clubs were organized in Fillmore County. There were further notices about bounties and many about collection of pensions. Finally, for the convenience of the veterans, there were medical examining boards, made up of local physicians, of the county bureau of pensions.

Through all the years, from the early fifties on down, there was constant promotion of railroads until finally the Southern Minnesota Railroad and the Caledonia, Mississippi and Western were established. Thereafter, in the late sixties, and as changes and extensions of the rails took place during the seventies, eighties and early nineties, there was increasing prosperity in some localities and there were declining fortunes in others. Many disappointed towns of Fillmore County were left to one side, especially by the Southern Minnesota Railroad. These little settlements, once thriving, founded each in the high hope that it would become the county seat, a shipping point or a manufacturing center like prosperous manufacturing towns in New England, were doomed to become "phantom villages;" a few of them were Beldena, Liberty, Hamilton, Washington, Etna, Carimona, Forestville, Fillmore and Elliota.

(To be continued in the April issue)

President's Letter

Before, during, and even since the war, European nations have borrowed and copied our industrial methods, which were recognized as superior. Now, the political forces of this country are trying to borrow and copy the equally inferior European medical system.

Such a statement as this raises two questions: First, how does the quality of medical care in this country compare with that of European countries having government-controlled medicine? And, second, how does the cost of medical care in this country compare with that of European countries having compulsory health insurance?

Before the war in Germany there was one physician for every 1,307 persons; in England, one to 1,069; and in the United States, one to 767. Up until 1936, for periods of three, five and ten years, heart disease, diabetes and cancer, respectively, increased at least 50 per cent more rapidly in both England and Germany than in the United States. Tuberculosis decreased about one-third in this country, whereas in England and Germany, only one-fourth and one-fifth, respectively. While diphtheria and other infectious diseases experienced a two-thirds decrease in this country, there was only a minimal decrease in England during the same years, and an actual increase of 200 per cent in Germany. Thus health conditions have steadily become worse in Europe.

In the United States, however, health conditions have constantly improved until they now are better than those of any other country with the same population in the world. Further evidence of the excellence of American medicine is seen in the fact that the United States is now the world's center for medical education. It has wrested this distinction from countries with compulsory health insurance.

Enough about quality, what about the cost? The National Resources Planning board found in 1935-36 that consumer expenditures for medical care amounted to less than four per cent of the national income. The Department of Labor, in 1940, stated that the average American wage earner spends $59 annually for his *family's* health; and, of this, only $13 goes to his doctor. And the Department of Agriculture found that families in small mid-western cities with incomes of from $1,200 to $1,750 spend only three per cent of income for physicians' and hospital bills.

Under the Wagner-Murray-Dingell Bill, a person receiving $100 a month pays $4 a month in taxes, while his employer pays another $4, making a total of $8 a month, or $96 a year. When the salary is $300 a month, the costs become $12 a month for wage earner and employer, or $288 a year. Thus, costs under this bill for compulsory health insurance are greater, both to individuals and to the country as a whole.

And what about costs of health insurance in European countries? Let it suffice to say that in Great Britain, in 1906, national and local taxes were equal to one-thirteenth of the national income; whereas in 1930, local and national taxes amounted to one-third of the national income. Furthermore, the cost of social (medical) service in Great Britain increased 1300 per cent during this period of twenty-five years.

From these incomplete data two conclusions can be drawn: first, that, beyond a doubt, medical care under the present American system is distinctly better than it is under the systems of England or Germany where compulsory health insurance obtains, and, second, that medical care under the present American system costs less than it does under the Wagner-Murray-Dingell Bill or under the system in England, where compulsory sickness insurance has existed since 1911.

Perhaps the inference to be drawn is that plans should be promulgated by the medical profession to provide adequate, high-standard medical care at low consumer cost. This is being done in Minnesota and nationally through the medium of voluntary, prepaid medical care projects. Thus advantages and benefits of both systems will be incorporated in the new American system.

President, Minnesota State Medical Association

Editorial

THE USE OF DEMEROL FOR OBSTETRIC ANALGESIA

THE use of demerol (1-methyl 4-phenylpiperidine, 4-carboxylic acid ethyl ester by diachloride) to relieve the pain of parturition was begun rather soon after the introduction of continuous caudal anesthesia. One of the most extensive experiences with this drug for the purpose under consideration, published thus far, is that of Schumann.[2] He reported the results of use of demerol and scopolamine in 1,000 deliveries at the Boston Lying-in Hospital. He declared that the primary purpose in his use of this type of medication was the establishment of complete amnesia. He concluded that this result was obtained in 70 per cent of the cases (uncorrected figure). He demonstrated to his satisfaction that the drug administered by the intramuscular or intravenous route, exerted no depressant effect on either the full term or premature infant. The only untoward effects on the mother were encountered among the group of patients who were given the drug intravenously. With the exception of transient nausea in a fourth of the cases and occasional vomiting no side effects were observed when the drug was administered slowly; that is, if a minimum of two minutes by the clock was taken to inject 2 c.c. of solution containing 100 mg. of demerol.

Irving,[1] also reporting from the Boston Lying-in Hospital, found that complete or nearly complete amnesia was experienced in 70 per cent of 2,446 obstetric patients in labor who received demerol, as compared to 85 per cent of 14,676 patients who received the barbiturates. In both series scopolamine was used concomitantly. Irving expressed especial concern over the development of maternal pulmonary edema. He reported thirty-five cases in the barbiturate group with three deaths or an incidence of one case of pulmonary edema in 419 deliveries. There were two cases of pulmonary edema among the 2,446 cases in which demerol and scopolamine were given or an incidence of one in 1,223 deliveries. Sixty-two per cent of the babies of the patients who received barbiturates breathed spontaneously (without increase in mortality) and 82 per cent of the babies of patients who were given demerol breathed spontaneously.

In one series, the results of which have not been published, demerol was used in about 1,500 obstetric cases. For a short time, early in this study, the drug was available for oral administration only. When given in this way demerol seemed to be disappointing as an analgesic. Administered intramuscularly, however, after a small initial dose of 1½ to 3 grains (0.1 to 0.2 gm.) of barbiturate, it gave fairly satisfactory analgesia in the great majority of cases. The dose was repeated every three to five hours; however, a total dose of 400 mg. or more was seldom used. On occasions when the first dose of 100 mg. had little effect in from thirty to sixty minutes, a second dose was effective. No untoward maternal side effects were encountered. The purpose of the medication in this series of patients was to give a reasonable degree of analgesia rather than complete amnesia.

No definite deleterious effects were noticed on the infants delivered after the use of demerol in this series. A peculiar type of asphyxia, however, occurred about five or six times as follows: The baby was born with good color and cried lustily at once but was found in a few minutes to be pallid and not breathing. Such babies responded in a few moments to administration of oxygen and gentle stimulation and were subsequently normal. There is no mention in the literature of demerol causing fetal asphyxia and the occurrences just mentioned in only about 0.3 per cent of the cases mentioned cannot be attributed directly to the drug.

Demerol alone does not seem to maintain relief of pain for patients who are in hard labor as well as colonic ether or morphine does, but it lacks the depressant effect of these drugs. The spasmolytic action of the drug noted by those who studied demerol pharmacologically is not impressive from casual observation of patients in labor who have received the drug.

All in all demerol seems to be a welcome addition to the armamentarium of the obstetrician for relief of the pain of labor, especially because it seems reasonably devoid of deleterious side effects to the mother and apparently does not depress too much the higher centers of either the mother or the child.

ARTHUR B. HUNT, M.D.

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MEDICAL PREPAYMENT PLANS GIVEN THE "GO" SIGN

THE House of Delegates of the American Medical Association at its December, 1945, meeting instructed "the Board of Trustees and the Council on Medical Service and Public Relations to proceed as promptly as possible with the development of a specific national health program, with emphasis on the nationwide organization of locally administered prepayment medical plans sponsored by medical societies." The term "specific national health programs" might have seemed rather ambiguous, but a "nationwide organization of locally administered prepayment medical plans" made it clear that the House of Delegates wants the prepayment plans on county or state basis to be pushed as fast as possible and their activities co-ordinated by a national organization.

The report of the outcome of the meetings held in Chicago beginning February 13, 1946, appeared in an editorial in the *Journal A.M.A.* for Februray 23, 1946 (p. 494). The editorial restates, in a revised version, the attitude and purposes of the organized profession, and explains what the AMA intends to do about prepayment medical plans in general.

Many members of the profession may have had in the back of their minds that one huge national prepayment medical insurance company was in the offing. This is not the case. What, in brief, has happened is that the Council on Medical Service and Public Relations has established a Division of Prepayment Medical Care Plans, appointed a Director and necessary staff, has drawn up standards for state or county medical care plans and is ready to assist local, county or state medical organizations in establishing local prepayment insurance units to which the Council is ready to give its seal of approval.

State and County Medical Associations have therefore received the word "go" and should take the necessary steps to promptly extend the number and scope of the already existent prepayment medical insurance organizations sponsored by medical societies.

It is well for medical men to bear in mind that prepayment medical insurance plans require the services of businessmen trained in the insurance business. Fortunately, they are ready to aid to the point of taking over on a non-profit basis.

A number of national insurance companies for some years have provided hospital and medical insurance mainly for large groups of employes. The overhead on the writing of individual policies of this sort is high. Even the Blue Cross hospital insurance is largely limited to groups of employes and the same is true of at least some of the medical insurance sponsored by the profession.

Coverage in general is limited to surgical and obstetrical care and, to some degree, medical care in hospitals. This is a good beginning but such insurance should be expanded not only to provide single policies but to include all possible illnesses, catastrophic in character, for the employe's dependents as well as himself. There is good reason to believe that such coverage can be made available at a cost attractive to people of low income.

REPORT OF DELEGATES TO THE AMERICAN MEDICAL ASSOCIATION

Chicago, December 3, 4, 5, 1945

The meeting was held at the Palmer House in Chicago, with 170 of the total number of properly credited delegates (175) present to make this one of the largest assemblies of representation the Association has had.

The report of the Council on Medical Service and Public Relations occupied fifteen pages of the program prepared for the AMA delegates. A supplemental report of equal size was submitted to the delegates during the session. Resolutions as approved by the House of Delegates will appear in the AMA *Journal*. The activities of the Council have been and will continue to be presented through the columns of *The Journal* and through the bi-weekly News Letter, which is mailed to AMA members desiring it.

Members of the Council have been studying problems confronting county and state medical societies, by holding sectional meetings with speakers chosen from local groups and from the central office. Dr. Joseph Lawrence, director of the Washington office, was included

at all of the sectional meetings so that he might report and advise on Federal legislation affecting medical service.

To further crystallize the wishes of the profession, two conferences were held immediately before the annual meeting of the House of Delegates. The executive officers of the state medical societies were invited to participate in a round-table conference held at the Headquarters Office. At the conclusion of each conference, resolutions were prepared with instructions that the Council on Medical Service and Public Relations submit them to the House of Delegates of the AMA. The Council complied with their instructions. There were three chief topics for consideration. The first dealt with Federal legislation. The resolutions favored an approval of the Hill-Burton Hospital Bill, which is to provide additional hospital beds where local need is indicated. The Pepper Bill was opposed since it plans to extend the Emergency Maternity and Infant Care program beyond the war emergency and is drawn to provide medical care for children until they reach the age of twenty-one years. Resolutions opposed the President's compulsory health insurance program and the new Wagner-Murray-Dingle Bill, on the grounds that it was the first step to federal regimentation of medicine, that the program would lower the quality of medical service and, further, that a voluntary plan of prepayment medical care on a competitive basis would preserve private enterprise, encourage investigation and raise the quality of medical service. A 14-point program as shown in the December 1 issue of the AMA *Journal*, page 945, was adopted.

The second topic dealt with resolutions recommending that the House of Delegates go on record as favoring a voluntary national prepayment medical service. The delegates approved the instructions which were given to the Council on Medical Service and Public Relations, with the advice of the board of trustees, that such a plan be initiated immediately. At present the Council on Medical Service is exploring several possibilities and is planning to integrate the present existing groups of voluntary prepayment medical service as a nucleus for the National plan. The national organization will then extend into states where new plans are contemplated or where plans do not exist.

The third topic dealt with public relations, an educational program to be extended throughout the country. The first step is to have physicians and surgeons familiarize themselves with the problems of the employer, the employed and the self-employed persons, like the farmer, to learn how better medical service may be given. This is to be done by talking with the patients and helping them to understand the costs involved in providing the modern advantages of medical science and to realize that a voluntary prepayment medical service is less expensive and offers a higher quality of service than the compulsory insurance. It was recommended that the information be extended to leaders of each community.

An address by Major General Paul R. Hawley on the care of the veteran was heard. Dr. Hawley included in his talk a discussion of plans for aiding veterans not only on the local scale but on the national level as well. His talk, which met with the approval of the entire house, appears in the AMA *Journal*, December 22, 1945, page 1192.

It was announced that the next meeting of the House of Delegates will be held in San Francisco July 1-5, 1946, and that hotel arrangements have been completed with the San Francisco hotel association. It is hoped that the fact that the war is over will bring about an increased attendance.

Since 1947 will mark the one-hundredth anniversary of the founding of the American Medical Association, elaborate preparations are being made for the 1947 meeting to be held in June of that year in Atlantic City, N. J.

One of the state delegates offered a resolution to the effect that the Council on Medical Service and Public Relations should be the sole spokesman for the American Medical Association. The resolution was not passed, since it was the opinion of the assembly that a lesser body in the association cannot assume the spokesmanship which rightfully belongs to the House of Delegates.

A new section, to be called General Practice of Medicine, and to be distinguished from the Internal Medicine specialty, was approved for addition to Scientific Assembly.

An important topic of discussion was the recent gift to the American Red Cross by the Army and Navy of 1,250,000 units of dried blood plasma which are being redistributed to all the states in the Union through the individual departments of health. Further discussion of this project will be found in MINNESOTA MEDICINE.

The Delegates to the American Medical Association wish to recommend that members follow carefully the Washington Letter and the Medical Legislation Program in *The Journal* of the AMA.

Results of the election of officers were as follows:

President-elect: Dr. H. H. Shoulders, Nashville, Tenn., Speaker of the House for the last two years.

Speaker of the House: Dr. R. W. Fouts, Omaha, Neb.

Dr. L. A. Buie has been elected to the Judicial Committee and Dr. A. W. Adson has been re-elected to the Committee on Medical Service and Public Relations.

It was learned at the meeting that Dr. George F. Lull, formerly deputy Surgeon General of the United States Army Medical Corps, has been appointed Associate General Manager of the AMA. He began work in his new position on January 1.

A. W. ADSON, M.D.
W. A. COVENTRY, M.D.
E. W. HANSEN, M.D.
F. J. SAVAGE, M.D.

Delegates to the American Medical Association

CANCER DETECTION CENTERS AND ALLIED CANCER PROJECTS

ARTHUR H. WELLS, M.D.
Chairman of the Committee on Cancer

At a recent meeting of the Committee on Cancer, the standards, rules, and regulations, and the means of establishing Cancer Detection Centers in Minnesota were considered—in line with the program approved by the American Medical Association at its House of Delegates meeting in December and by the Council of the Minnesota State Medical Association at its December 16th meeting. This program is a co-operative venture between the Minnesota Cancer Society and the Minnesota State Medical Association and its component county societies, the Cancer Society underwriting the cost of the clinics where desired, and the medical profession initiating, directing and staffing the clinics. Increasingly large sums of money are being collected by the Minnesota Cancer Society, allotments of which are being made available for County Medical Society cancer projects.

For many years the activities of the American Cancer Society were largely limited to a program of cancer education and cancer research. However, it became increasingly apparent that this program was not complete, and that there was a great need for service to the patient. There developed, as a recognition of this need, the formation of the Service Division of the American Cancer Society. This Service Division is governed entirely by practicing physicians, with representatives from the American Medical Association, the American College of Surgeons, and the American Roentgenological Society. The Service Division is responsible for all projects concerning the patient as a patient, and includes the following specific functions: (1) survey of needs, (2) detection, prevention, or well-person clinics, (3) diagnostic clinics, (4) treatment centers, and (5) care of advanced cancer patients. The American Cancer Society, or its State or local divisions, may not own or operate any clinic, laboratory, hospital, or other facility for the care of cancer patients. The Cancer Society is a benevolent pressure group acting under the constant advice of physicians. All cancer projects financed or aided by the Cancer Society must have the approval of the County and State medical groups.

Three sets of standards, rules and regulations are to govern the establishment and maintenance of Cancer Detection Centers: (1) those of the American Medical Association, (2) those of the State Cancer Committee, and (3) those developed by local County Medical Societies.

The following American Medical Association principles of operation of Cancer Detection Centers are reviewed:

1. Definition: A cancer detection, cancer prevention, or well-person clinic is designed to defect abnormalities, not producing symptoms sufficient to send the patient to the doctor. These clinics do not diagnose or treat diseases.
2. No such clinics shall be established in any community without the approval of the County Medical Society.
3. The examination shall be as complete as local facilities will permit with the exception of biopsy. In all instances this examination shall include a complete history and physical examination.
4. Records may be as complete as desired. They shall in all instances include the abnormal findings.
5. If abnormalities are detected, the patient shall be referred to the family physician for diagnosis and treatment. If there is no family physician, the patient shall be referred to a physician or clinic selected by the patient from a group chosen by the County or State Medical Society. Such reference shall include a complete report of the screening examination.
6. One month after such reference to a physician, the case shall be followed up by letter or social service visit and a complete report of the diagnosis and treatment shall be obtained.
7. Cancer Detection Centers must be located in hospitals approved by the American College of Surgeons.[1]

The policies recommended by the State Committee on Cancer follows:

1. A Cancer Committee shall be appointed in each interested County Society.
2. The Chairman of the Cancer Committee of each County Society shall be charged with the responsibility of interesting hospital authorities and hospital medical staffs in his community in setting up clinics. He is also responsible for maintenance of standards in established clinics.
3. The hospital staff shall select a special committee to be responsible for setting up the clinic—to determine how much space is needed, how much money from the Minnesota Cancer Society will be needed to finance the project, to select the personnel and physicians to staff the clinic and to assign their duties.
4. The Minnesota Cancer Society shall provide uniform medical history and physical examination forms to be drafted by the State Committee on Cancer for the use of examining physician. This Society shall also provide advertising, clerical help, and telephone facilities for aiding in arrangement of appointments, receptionists, and the maintenance of records, as requested and approved by the County Medical Society or its Cancer Committee.
5. The local County Medical Society must approve of every clinic before it is established.
6. Clinics shall be free to all persons coming in for examination.
7. A nurse may be employed to take the preliminary case history, but this must be checked and enlarged upon later by a physician.
8. An internist and a surgeon shall examine each individual patient for cancer, its precursors, or other ailments, with particular attention to the more common and accessible sites of cancer.
9. Physicians shall donate their services without charge.

At the outset, it is hoped that trial clinics will be set up in the three largest cities: Minneapolis, Saint Paul and Duluth. However, as soon as it is practical and

(Continued on Page 293)

MEDICAL ECONOMICS

Edited by the Committee on Medical Economics
of the
Minnesota State Medical Association
George Earl, M.D., Chairman

ORGANIZED AGITATION FOR SOCIALIZED MEDICINE TOUCHED OFF AT CONFERENCE

Using Minnesota as trial ground, proponents of a program of federally-administered compulsory health insurance got together for a "Health Workshop" Conference in St. Paul at the Lowry Hotel February 6 through 9. At closed sessions leaders in the National Farmers Union, the American Federation of Labor, the Congress of Industrial Organizations and the Railroad Brotherhoods, met to find the most effective way of spreading the gospel of national health insurance.

Delegates were invited in from Minnesota, Wisconsin and the Dakotas to "unite and demand the political action necessary to improve this nation's health care and standards."

Conference Turns Out "Technical Consultants"

During the four-day sessions, confined to representatives of the sponsoring unions, the delegates were briefed and provided with folios of information so that they might go back to their home states as "technical consultants" and set up interim committees for the purpose of educating the people to the need for improving health facilities. It is hoped apparently that as a result of a carefully planned campaign of education, the program for socialized medicine might gain favor and that more people would put pressure on elected officials to support legislative measures of the Wagner-Murray-Dingell brand. It was decided at the meeting that there will be a series of follow-up meetings, comparable to this one, which will take in more localized groups.

Some of the speakers were loudly critical of the medical profession; others tempered their comments. A couple of the speakers were outspoken in their praise. It was voted to invite the representatives of the medical, dental and allied health professions to sit in and be heard at the follow-up meetings in the various states.

National Farm Union President Sounds Keynote

James G. Patton of Denver, president of the National Farm Union, made the keynote address on the evening of the eighth. His punch lines were that President Truman's five-point program, submitted to Congress last November, was fine, but not drastic enough, and that it was high time the "working" farmers and "working" laboring men took political action to insure a "progressive program across the board to guarantee every man, woman and child needing it a free education, free medical care and a decent standard of living."

Obstructionists, such as congressmen and governors, who are against social legislation, said Mr. Patton, must be done away with. Throughout his talk, Patton stressed that the program being launched at the conference was to be one of action. "The lack of medical care," he declared, "is going to be ended *now* and not 25 years from now!"

This health workshop conference was planned back in June of last year, when Mr. Patton and Surgeon General Thomas Parran met at Washington. At that time the facilities of the U. S. Public Health Services were offered to further a universal program for adequate health care for all people with the emphasis on those in the $1,000 or less income bracket, *provided* that other federal agencies in the health field would also cooperate in the venture.

Present at the health workshop conference to help work out the details of the health education program were specialists from various governmental agencies, including the Department of Agriculture, the U. S. Public Health Service, the Federal Housing Agency, the Social Security Administration and the Department of Labor.

SOCIAL SECURITY COSTS IN U. S. RISING RAPIDLY

With the government providing more and more services to citizens, the cost of the social security program in this country is rising steadily. Not that this increased spending is all the result of an actual improvement in services offered—the lion's share of the social security budget is being turned over to the thousands of employes in the bureaucratic honeycomb.

The latest service which some would delegate to the government is that of providing health insurance, such as is called for in the Wagner-Murray-Dingell Bill. And although no tax provisions are emboided in the context of the bill, it stands to reason that the program will have to be financed through an additional tax burden on the American people, all of which will contribute another substantial hike to the cost of social security.

This is the social security picture today—a large and ever-growing financial drain on the budget by bureaucrats, plus the prospect of a large additional drain.

According to conservative estimates, it would take at least 600,000 additional salaried government employes to administer the compulsory health insurance program of the Wagner-Murray-Dingell Bill. This army of bureaucrats will draw on an average of $3,000 a year each, according to the present average federal salary scale.

According to Representative Charles W. Vursell (Illinois, Republican), there are now a total of 1,141 bureaus in the federal government, which employ 3,649,383 persons. By adding the salaries of the workers who will be needed to manage a federal health insurance program to the present huge payroll, an estimate can be obtained of what the total costs will be should the Wagner-Murray-Dingell Bill become law.

HEALTH BENEFIT PROGRAMS ESTABLISHED THROUGH COLLECTIVE BARGAINING

During recent years, an increasing number of unions are having health benefit plans included in the terms of their agreements with employers. A report of this development, summarized in the August, 1945, *Monthly Labor Review*, published by the Bureau of Labor Statistics of the U. S. Department of Labor, indicates a significant trend in labor circles toward making medical coverage a part of negotiated contracts. Some of the more recent health benefits have been negotiated in lieu of wage increases which could not be obtained under the wartime wage stabilization program.

Details of three types of health benefit programs, determined by their method of administration, are set forth in the article: those administered entirely by the unions, those administered jointly by union and employer, and those administered through private insurance companies with the insurance company assuming responsibility for eligibility of claims and payment of benefits.

In the main, health benefit plans provided under union agreements include weekly disability benefits usually ranging from 50 to 60 per cent of regular earnings; many plans provide surgical benefits ranging from $100 to $175; and hospital expenses ranging from $4 to $5 a day for 31 days, are usually allowed for any one continuous disability. A few plans allow specified payments for doctors' services up to a maximum of 50 visits for any one disability, which usually begins with the first treatment in the case of accident and the fourth, in case of illness. As might be expected, benefits tend to be higher under plans negotiated in industries having relatively high wage scales.

According to the article, the enlarging of health-benefit programs to cover more workers and provide more services, and the solving of administrative difficulties, such as the problem of the length of time to be covered during temporary lay-offs, seasonal slack periods and leaves of absence, remain to be worked out in future negotiations.

AMERICAN ACADEMY OF PEDIATRICS EVALUATES PEPPER BILL

After carefully evaluating the Pepper Bill, the "Maternal and Child Welfare Act of 1945," which would provide grants by the federal government to the states for providing medical services to mothers and children (up to the age of 21), the American Academy of Pediatrics has adopted a resolution setting forth specific criticisms of the bill.

The first criticism which the Academy notes is that the bill as now written states that services and facilities furnished under the state plans are to be available to all mothers and children who elect to participate in the benefits and therefore denies the states' rights to determine eligibity.

The Academy of Pediatrics does not favor the use of federal funds for those able to provide medical care from their own resources.

The Academy notes further that the bill excludes fee-for-service as a means of paying practitioners for their services, makes inadequate provision for paying groups of physicians or institutions for professional services and does not prevent professional personnel groups or institutions from accepting supplemental payments.

Issue is taken with the endorsement by the bill of the Children's Bureau as the administrative agency of the National Health program, as outlined, without the proper assurance of the program's integration with other health activities of the federal government.

Issue is taken also with the definition of federal or state advisory committees as regards personnel, method of appointment, advisory and policy-making roles or manner of giving authority to the record of consultations with and recommendations to the administrators.

It is noted that no provisions are made for varying the remuneration for services according to the differing costs from state to state; nor is there any provision for the prevention of arbitrary requests on short notice by the federal administrative agency for reports from the state health agencies or from the practitioners. The provisions for handling claims are unsatisfactory as are the specifications regarding adequate dental care.

Research and Education Not Provided For

The Academy feels that one of the most serious errors of the bill is that it fails to make adequate provision for the protection of the well-organized teaching services and for the support and encouragement of research pertaining to the improvement of maternal and child health services and medical care. On these, the Academy states, the future of the quality of medical care is dependent.

The final criticism is aimed at the lack of assurance in the bill that state plans shall not expand too rapidly for the available administrative and professional personnel and resources. For these reasons, the Academy concludes in its resolutions, the Maternal and Child Welfare Act of 1945 does not represent the best forms of legislation for the purpose for which it was written.

For those considering proposed legislation, the Academy directs attention to its fact-finding study of child health services, now in progress, which at its conclusion, should assist in the development of sound programs at state levels based on demonstrated needs.

Pending the completion of this study, it is recognized that urgent need exists in some states that should be met immediately. To this end the Academy recommends that additional federal funds be made available for grants-in-aid to the states under existing Maternal and Child Health and Crippled children's programs under the Social Security Act.

FELLOWSHIP GIFT RECEIVED

A gift of $25,000 to the University of Minnesota Medical School has been received. The money is to be used to provide five graduate medical fellowships of $1,000 a year each, covering a five-year period.

Veterans of World War II are to receive the fellowships, which may be used for study in any recognized field of medicine.

AERIAL CLINIC IN ALASKA

In Alaska where "everybody flies" among the air-age projects under way is the establishment of an aerial medical clinic. Doctors and dentists are outfitting a plane with x-ray equipment and equipment for minor surgical work. The medical men don't intend to perform major operations on the spot; instead, when necessary, the patients are to be flown to the nearest town with hospital facilities.

MINNESOTA STATE BOARD OF MEDICAL EXAMINERS

230 Lowry Medical Arts Bldg., Saint Paul, Minnesota

Julian F. DuBois, M.D., Secretary

TO THE MEMORY OF MAX WILLIAM ALBERTS, M.D.

We were all shocked to learn that one of our members, Dr. Max Alberts, died suddenly at the age of forty-five from a coronary lesion, on June 11, 1945. Though we were aware of the fact that he had been annoyed by precordial distress, none of us appreciated his critical condition, nor did Doctor Alberts permit himself to be an invalid. On the fatal day, in spite of pain, he reported at the hospital, performed a laparotomy and succumbed on his way home while driving his car.

Max, as we all knew him, had served with us as a member of the State Board of Medical Examiners for a period of eleven years, beginning his service on May 9, 1935. During this time we had learned to know him, both professionally and socially. Professionally he had

made satisfactory progress. He was liked by his colleagues and developed an enviable surgical practice. His decisions on the Board were fair. He showed his understanding of human frailties by being inclined to soften or lessen the maximum penalties for violators of the medical codes and laws.

Max gave generously of his time to medical affairs and served on numerous city, county and state committees. He took his professional work very seriously, but he did enjoy relaxing. He appreciated his social contacts, enjoyed discussing sports and politics, on both of which subjects he was well informed.

Though Max's demise is our loss, it is not comparable to the loss experienced by his wife, son and daughter. We have lost a colleague, a regular guy whom we shall always remember; they have lost a husband and father who loved his family and whose family loved him. Max has not lived in vain.

MINNESOTA STATE BOARD
OF MEDICAL EXAMINERS.

Minneapolis Ex-convict Sentenced to One Year Term for Violating Basic Science Law

State of Minnesota vs. Harry C. Chappell

On February 8, 1946, Harry C. Chappell, forty-eight years of age, 2239 Cole Ave. S.E., Minneapolis, was sentenced by the Hon. Arthur W. Selover, Judge of the District Court, to a term of one year in the Minneapolis Workhouse. Chappell was sentenced for representing himself as a doctor in violation of the Minnesota Basic Science Law.

Chappell was arrested on February 6, 1946, by Detectives Lillejord and Henseler of the Minneapolis Police Department following an investigation by Inspector Bernath and Captain Mullen of the Minneapolis Police Department, and representatives of the State Board of Medical Examiners. Chappell, who has no medical training of any kind, had telephoned at least two women who had inserted advertisements in the Sunday *Minneapolis Tribune* of February 3, 1946. The women were seeking stenographic work and Chappell, in answering the advertisements, stated that he was a doctor and that he was about to open an office in the Masonic Temple Building, Minneapolis. One of the applicants went to Chappell's home address where he insisted that she submit to a physical examination as a prerequisite to being given employment. The applicant objected to a physical examination on the ground that she had been examined by her family physician in December and found to be in good health. Chappell insisted on the physical examination and had the applicant remove her clothing. Following the examination the applicant informed her parents of what had taken place and the matter was immediately reported to the Hennepin County Medical Society and to the Minnesota State Board of Medical Examiners.

Chappell has a long criminal record dating back to June, 1922, at which time, he pleaded guilty in the District Court of Hennepin County to robbery in the third degree. For that crime Chappell served a term in the St. Cloud Reformatory. In May, 1930, Chappell was sentenced to a five-year term in the North Dakota State Prison for burglary in the third degree. He served three years and three months of that sentence. In March, 1934, Chappell was sentenced to a term of two to fourteen years in the District Court of Ramsey County, Minnesota, for the crime of indecent assault. Chappell served nine years of that sentence.

In June, 1944, Chappell was sentenced in the District Court of Hennepin County to a term of one year in the Minneapolis Workhouse for attempted carnal knowledge involving a fifteen-year-old girl. Chappell stated, when questioned by Minneapolis Police officers, that his only occupation outside of serving more than twenty years in various prisons, was that of a bell-hop at several Minneapolis hotels. Chappell admitted that he had no intention of attempting to practice medicine and attributes his present difficulty, as well as two of his previous encounters, to "liquor and sex."

Michigan Physician's Minnesota License Revoked

Revocation of the License of Elmer J. Hagenbaugh, M.D.

The Minnesota State Board of Medical Examiners, at its regular meeting on February 8, 1946, revoked the license to practice medicine held by Elmer Jasper Hagenbaugh, M.D. Dr. Hagenbaugh was convicted on May 10, 1945, of the crime of manslaughter, in the Circuit Court of Calhoun County, Michigan. The criminal charge followed a criminal abortion performed by Dr. Hagenbaugh. Dr. Hagenbaugh was sentenced to a term of five to fifteen years in the Michigan State Prison at Jackson, Michigan, and is serving his sentence at the present time. Dr. Hagenbaugh was found guilty by the Board of immoral, dishonorable and unprofessional conduct in connection with the Michigan offense.

Dr. Hagenbaugh was practicing at Athens, Michigan, at the time of his conviction. On August 11, 1944, Dr. Hagenbaugh was convicted in the Superior Court of Elkhart County, Indiana, of the crime of abortion and was sentenced to a term of not less than three, nor more than fourteen years in the Indiana State Prison and fined the sum of $1,000. Upon the payment of the fine the Court suspended the prison sentence. Shortly thereafter Dr. Hagenbaugh moved from Elkhart, Indiana, to Athens, Michigan.

Dr. Hagenbaugh was born at Sherwood, Michigan, in 1864. He graduated from the Toledo Medical College in 1905, and was licensed in Minnesota, in 1909, by reciprocity with Illinois. Dr. Hagenbaugh practiced medicine for a short time in 1909, at Rochester, Minnesota, and then moved to Elkhart, Indiana.

License of Minneapolis Physician Suspended for Five-Year Term

Revocation of the License of Harold E. Harbo, M.D.

On February 8, 1946, the Minnesota State Board of Medical Examiners suspended, for a period of five years, the license to practice medicine held by Harold E. Harbo, M.D., who formerly maintained an office at 4 West Lake Street, Minneapolis. Dr. Harbo was found guilty by the Board of immoral, dishonorable and unprofessional conduct in connection with an abortion performed on a thirty-two-year-old unmarried Minneapolis woman in April, 1945.

An investigation was commenced by the Minnesota State Board of Medical Examiners when it was learned that the patient was hospitalized with an infection following the alleged abortion. The patient gave a statement in which she claimed that she consulted Dr. Harbo for the purpose of having him perform an abortion upon her and that he advised her to get a room and that he would have someone come to the room for the purpose of doing the abortion. The patient stated she paid the sum of $150 but declined to identify Dr. Harbo as the person who came to her room and performed the abortion. Dr. Harbo appeared in person before the Board and admitted that he performed the abortion.

Dr. Harbo was born at LaCrosse, Wisconsin, in 1892. He graduated from the University of Minnesota in 1921 with a Bachelor of Medicine Degree. He received his Doctor of Medicine degree in 1922. He was licensed in Minnesota by examination in 1921.

Minnesota Academy of Medicine

Meeting of December 12, 1946

The regular monthly meeting of the Minnesota Academy of Medicine was held at the Town and Country Club on Wednesday evening, December 12, 1945. Dinner was served at 7 o'clock and the meeting was called to order at 8:15 by the President, Dr. A. G. Schulze.

There were forty-six members and two guests present.

Dr. E. M. Jones read the following memorial to Dr. Max Alberts and a motion was carried that this be spread on the minutes of the Academy and a copy sent to Dr. Alberts' family.

MAX WILLIAM ALBERTS
1899—1945

Max William Alberts was born at Berlin, Wisconsin, October 28, 1899. He attended the public schools at Berlin, Wisconsin, before entering Ripon College where he received his preliminary college work. He entered the Medical School at the University of Minnesota and received his degree in Medicine in 1923.

He served his internship at St. Joseph's Hospital of St. Paul, Minnesota, and on completion of this service, became associated with Dr. W. C. Carroll in Saint Paul as surgical assistant.

In July 1925, he married Lola Schultz, who, with two children, a son, William, and daughter, Mary, survive.

Dr. Alberts always exhibited a most sincere interest in Medicine and took an active part in the proceedings of the medical societies to which he belonged. He was a member of the American Medical Association, the Ramsey County Medical Association, and the Minnesota State Medical Association. He was also a member of the Western Surgical Association, the American College of Surgeons, the Minnesota Academy of Medicine, and the St. Paul Surgical Society. He was a member of the Minnesota State Board of Medical Examiners, a clinical assistant in Surgery on the Medical Staff of the University of Minnesota, and was on the Board of Trustees of Ripon College.

Dr. Alberts had no particular hobby, but he was very much interested in sports, especially baseball and football, and had many close friends among the coaches and players.

Dr. Alberts was a tireless worker, and was always willing and ready to lend a helping hand when necessity arose. His confreres in St. Paul and the members of the Minnesota Academy of Medicine have lost a good friend and a good surgeon.

The election of officers followed and the following were elected to serve for the year 1946:

President ..... Dr. S. E. Sweitzer, Minneapolis
Vice President ..... Dr. E. M. Hammes, Saint Paul
Secretary-Treasurer .... Dr. A. E. Cardle, Minneapolis

The scientific program followed.

THE SIGNIFICANCE OF BLEEDING FROM THE RECTUM

JAMES K. ANDERSON, M.D.
Minneapolis, Minnesota

Bleeding from the rectum is one of the commonest complaints presented by the patient consulting the proctologist. Mummery,[4] in various articles, states that bleeding may result from almost any bowel complaint, and that mistakes and difficulties in diagnosing the source of bleeding from the rectum are many; in fact, more frequent than in other forms of hemorrhage. To the patient, bleeding from the rectum usually means one or two things—hemorrhoids and cancer. Too often the diagnosis of hemorrhoids is accepted by the physician without further investigation and this is one of the common pitfalls in diagnosis or lack of diagnosis that we see in malignant lesions of the rectum and rectosigmoid. A noticeable number of individuals with malignancy coming into our office have been subjected to a hemorrhoidectomy during the preceding few weeks or months, because of bleeding from the rectum; these patients had hemorrhoids as well as another lesion.

Sources

The sources of blood passed by rectum are multiple as well as the lesions responsible for the bleeding.

Any ulcerative, proliferative, or inflammatory lesion of the mucosa of the bowel may be the source of the blood, and blood may come from any portion of the alimentary tract. It is fairly easy to distinguish blood coming from the small intestine, as this blood is partly digested in the intestine and will be dark or have the ordinary characteristics of melena, although I have seen two patients with severe hemorrhage from the rectum from a gastric or duodenal ulcer, where the blood passed was bright red and in bright red clots, having come through the entire tract in a very short time. Blood coming from the large intestine is not so easily diagnosed as it is often little changed in characer and may be confused with blood originating in the rectum. As a rule, however, it is mixed with stool and mucus and is not so bright. Any blood remaining long in the colon or rectum becomes dark.

Blood coming from lesions in the rectum, carcinoma, polyps, ulcers, internal hemorrhoids, is usually bright red and not mixed with stool. Hemorrhoidal bleeding usually comes with and after the stool is passed, but internal hemorrhoids may be irritated or abraded by the passage of the stool and this blood accumulates in the rectal ampulla to be passed some time later in a changed, clotted and darkened form and so suggests blood from higher in the tract.

Thesis read before the Minnesota Academy of Medicine.

Severe hemorrhage from diverticulitis is fairly common. In my experience, the blood passed is thin and dark, not clotted, and in some quantity. These hemorrhages come suddenly and may be serious but usually are self-limiting and come in attacks. At times the bleeding is preceded by dull colonic pain. These patients are usually over 40 and obese. Diverticulosis occurs in about 5 per cent of individuals over 40.

Nothnagel's[8] table, made many years ago, gave the common causes of rectal hemorrhage as: hemorrhoids, dysentery, typhoid and carcinoma. He stated that small quantities of pure blood either light or dark suggested the rectum or sigmoid as the source. When blood forms a coating on the stool, the cause is in the lower part of the bowel. On the other hand, if the blood is mixed with the stool, the source is probably high and from the small bowel.

Landsman[9] states that the presence of a constitutional disease of some nature has a decided bearing on the possibility of bleeding from the rectum, notably tuberculosis, syphilis and hepatic disease. Primary tuberculosis is very rare, but secondary infection from the lungs is common. In 1230 autopsies in the Royal Victoria Hospital, but two gave evidence of primary involvement in the bowel, while 285 were secondary to tuberculosis elsewhere. Syphilis may involve the lower portion of the intestinal tract as chancre, or in its secondary or tertiary manifestations as mucous patches, condylomata, fissures, ulcers, proctitis, gummata, and strictures, besides congenital syphilitic lesions principally of the anus which give rise to ulcerations. Of these, chancres and gummata are infrequent; the others are fairly common. Hepatic disease, though rare, may be a fruitful source of rectal bleeding because of its mechanical and other effects in bringing about pathological conditions of the rectum.

Hence, the character of the blood expelled is not so important as was formerly taught. Merely because the patient loses blood from the rectum is no proof that the source of it is in that organ. It may come from any part of the intestinal tract and its color is by no means infallible evidence of its origin despite the fact, which is often accepted, that if the blood is dark it comes from high up, whereas if it is red, the lesion which causes it is low down. Blood coming from high up is indeed dark, clotted and otherwise altered, but only if retained in the bowel a sufficient time. If it is expelled before these changes have had time to occur, it will be bright red. Conversely, blood from low down may be retained in the rectal ampulla long enough to become dark and possess all the characteristics of blood coming from higher up. We may even have at times both liquid and clotted blood from the same lesion at the same time, whether situated high or low, thus tending further to confuse the issue. So, we cannot draw any positive conclusions from this circumstance until the findings have been confirmed by more reliable data, a careful history, and proctoscopic and x-ray examinations.

Postoperative bleeding or hemorrhage following anorectal surgery occurs in a small percentage of cases. This is usually due to a slipped suture if within twelve hours, but may occur up to fourteen days due to slough and vessel erosion, and prove embarrassing. The diagnosis and treatment is evident.

Chronic ulcerative colitis is one of the common lesions producing bleeding. This bleeding may be slight or profuse, is usually thin and mixed with stool and mucus, and accompanied by frequency of movements. Amebic and bacillary dysentery are also seen and give somewhat the same symptoms. Factitial proctitis is commonly seen following radium implantation in the cervix. This lesion is seen on the anterior rectal wall as a telangiestasis. The bleeding from this lesion may be brisk but is usually noted in small amounts in the stool.

In cases of hemorrhage from the rectum, the expelled blood should be examined soon after its passage, if possible, and before further chemical changes have occurred. This may give some indication of the source.

Quantity

The quantity of blood lost depends in great measure upon the size of the lesions, its character, and the extent of the destructive process. Carcinoma of the colon or rectum, multiple polyposis, ulcerative colitis, the dysenteries, and diverticulosis, show extensive areas of involvement and the pathological process inclines to deep destruction rather than to spontaneous recovery; hence, bleeding from such sources is naturally more severe and persistent, and the expelled material more often contains an abundance of mucus, pus, and pieces of tissue, in contradistinction to such innocent causes as hemorrhoids, single polyps, prolapse, et cetera, in which the blood will be found to be unmixed and show no products of tissue destruction. These lesions may also cause interval bleeding. It may be accepted, then, in a general way, that mixed blood points toward serious conditions, though the converse of the proposition does not invariably hold true, for unmixed blood does not necessarily predicate benign lesions.

When there is any pathological condition of the bowel which has a tendency to bleed, any increase in the intra-abdominal pressure, due to straining, vomiting, or defecation, may initiate it. However, the sudden appearance of a large hemorrhage, without any previous history of rectal disturbance and independent of any bowel movement, is quite characteristic of some cases of carcinoma of the rectum. One occasionally obtains a history of a similar onset in hemorrhoids or polyps, but close questioning will nearly always reveal a distinct interval history of rectal symptoms preceding the hemorrhage.

Perverted function of the bowel and any co-existing bleeding have a definite relationship to each other. When there is persistent constipation, the force and straining required to expel the stool are liable to cause injury to the mucous membrane, aside from any medical or chemical means used to help secure an evacuation. In an impaction the same factors operate with the further addition of a possible necrosis due to pressure of the hard, dry fecal masses on the delicate mucous membrane. When the stools are abnormally frequent, they are likely to contain blood from the lesion which causes the diarrhea, if not from the straining and tenesmus set up by the discharges.

Zobel[6] states that bleeding from gastric and duodenal ulcers and cancers is the usual cause of tarry stools. But these may also follow severe hemorrhages from the nares, lungs, esophageal varices, or typhoid ulcerations. Portal obstruction from any cause, acute yellow atrophy of the liver, purpura, hemophilia, leukemia, aneurysm, mesenteric thrombosis and scurvy may also be causes. Carcinoma of the colon seldom produces profuse hemorrhage and hence not tarry stools, but gross and occult blood.

Profuse bleeding originating in the rectal ampulla is usually from an ulcerating carcinoma of the rectum or rectosigmoid, a detached polyp, or sloughed hemorrhoid. The passed blood is bright and may contain clots mixed with stool.

There is a group of lesions and diseases of particular interest to the internist in which there is bleeding from the rectum in varying amounts and color, either free or mixed. They are: gastric and duodenal ulcers, yellow fever, septicemia, endometriosis with rectal involvement, pyemia, malaria, dengue, typhoid, jaundice, abdominal injuries, intestinal hemorrhages not due to tuberculous ulceration but to tuberculous pulmonary disease, sudden diarrheal attacks with bloody mucus which occur in exophthalmic goiter, arsenic and phosphorus poisoning, cancer of the small intestine, intestinal parasites, sclerosis of the intestinal vessels, chronic nephritis and acute enteritis.

The most common causes of bleeding from the rectum in children are polyps, prolapse and anal fissures, although well-developed hemorrhoids are occasionally seen. Proctitis, colitis, and intussusception also cause bleeding. Blood and mucus passed by a child under one year of age is almost diagnostic of an intussusception. Foreign bodies (thermometers, pencils, et cetera), introduced into the anus, are also fairly common causes of trauma and resulting bleeding. Meckel's diverticulum must also be considered, the blood in this case being dark and mixed with stool.

Diagnosis

The diagnostic problem involved in bleeding from the rectum is based on an adequate history and careful anoscopic, proctoscopic, and roentgenologic examination.

I feel that the examination should begin from below, with a digital examination followed by a short anoscope—the type with which the physician is familiar; then the electrically lighted proctoscope inside for at least 20 to 35 cm., if possible, examining every inch of the mucosa and looking behind the valves of Houston. In many cases, due to angulation or spasm, it is impossible to examine farther than 16 to 18 cm. without anesthesia. If blood is seen to be coming from beyond the proctoscope, or no source of bleeding is found for the distance examined, a barium enema is indicated which may reveal polyps, carcinoma, or diverticulosis. The openings of rectal and low sigmoidal diverticula are occasionally seen. If proctoscopic examination and barium enema are negative, a gastro-intestinal study is necessary. X-ray examination for carcinoma of the rectum is often misleading. These instrumental examinations are not difficult if proper equipment is on hand for the removal of blood and any liquid stool found in the bowel, and certainly should be done where any blood is passed. The more common cases of bleeding will be diagnosed and the more obscure ones will be gradually approached by the process of elimination. Individuals with severe hemorrhage may be proctoscoped at once if the general condition permits, but the roentgen examination may have to be postponed until the bowel can safely be prepared properly by catharsis or enemata. Fortunately, in most of these cases the bleeding will stop of its own accord and in a few days will permit a complete study. Recurrence, of course, is most likely.

I would like to cite a case of a rather unusual type of rectal hemorrhage which came under my care.

This man, aged forty, a traveling salesman, was out in the western part of the state. Following a bowel movement he passed considerable bright red blood and reported to a local physician who examined him and gave him some suppositories. He started to drive east and at several towns stopped at toilets and each time passed only bright blood in some quantity. He also visited several other physicians who gave him similar treatment.

I saw him about six hours after the bleeding had started and he again had a rectum full of bright blood. This was removed by suction and I could see the stump of a polyp in the rectal ampulla with a pulsating vessel. On close questioning, he stated that he had felt something protrude from the anus at the time he had first noted the blood and when he wiped himself he felt something snap and noted a piece of meaty tissue on the paper. The pulling off of a pedunculated polyp or adenoma may often happen, but this is one case that was very definite and might have proved tragic.

I would also like to report one typical case of severe hemorrhage from the bowel as the first evidence of a rectal carcinoma.

This man, aged fifty-seven, on September 18, 1943, had three severe hemorrhages from the rectum. These, of course, frightened him and he reported for examination. On close questioning, he admitted that for several months he had noted a red stain in the toilet bowl but never any gross bleeding. There had been no weight loss, but some bowel habit change. He also knew that he had hemorrhoids and had considered these the source of the bleeding.

Treatment

Surgical treatment is indicated in the majority of these cases to remove the source of the bleeding, whereas others require medical treatment, rest and diet.

Summary

In summary, I would say that bleeding from the rectum is a common symptom. Lesions and diseases are multiple. Diagnosis may be evident or very difficult to establish. Careful history, ocular and x-ray examinations will usually reveal the cause. Hemoglobin estimation will reveal the extent of the blood loss.

A plea is made for careful proctoscopic examination of the rectum and rectosigmoid, especially when hemorrhoids or evident anal pathology which commonly causes bleeding exists, and a complete gastro-intestinal

study with the x-ray when lesions are not found by ocular examination.

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Discussion

DR. L. G. RIGLER, U. of M.: It may seem paradoxical for a roentgenologist to agree with Dr. Anderson about the lack of accuracy in the roentgen examinations of the rectum. I am amazed at how many patients are sent in for a barium enema study without previous proctoscopic examination when a lesion in the rectum is suspected. It is an extremely difficult thing to demonstrate such lesions and roentgen diagnosis is in doubt because this is one portion of the colon where a good filling is not obtained. There are, of course, a few cases in which proctoscopy and sigmoidoscopy are impossible and there even rectal examination is doubtful, but aside from these, when one is dealing with a lesion within reach of the proctoscope or the finger, I think it is better to get the direct determination of the lesion rather than by an indirect procedure such as with the roentgen examination.

Determination of a source of bleeding from the gastro-intestinal tract is one of the most difficult things we have to encounter. I don't know whether it is commonly realized how many patients there are in whom the source of bleeding is never ascertained and that includes the autopsy examination. One of these sources which is difficult to explore is the small intestine and lesions, therefore, are commonly overlooked in this area. In those cases in which there is persistent blood and all of the usual examinations are negative, including x-ray studies of the stomach and duodenum and colon, a very vigorous effort to investigate the small bowel should be made. I am sure that some of my most flagrant errors have been in this field of endeavor. In recent years we have been using the so-called small intestinal enema —a tube is introduced into the duodenum and 1000 c.c. of barium mixture is quickly put into the small intestine through this tube. A sufficient distension and visualization of the small bowel is produced in this way so that gross lesions at least can readily be determined.

I am sure that every radiologist has a feeling of comfort about the lowel bowel if there is a proctologist available so that he can determine some of the things which it is difficult to elucidate by x-ray examination.

DR. E. M. JONES, Saint Paul: A few years ago I saw a man forty years of age, who presented a clinical picture typical of acute diverticulitis of the sigmoid. He had a temperature of 101°, leukocytosis, with pain and tenderness in the lower left abdomen. He also had rectal bleeding. He was hospitalized, and after a few days an x-ray examination of the colon revealed diverticulitis of the sigmoid.

About a week after admission, a proctoscopic examination was made, and no source of the rectal bleeding was found. The patient insisted on leaving the hospital, but he was advised to return for another x-ray study of the colon.

A few months later I was called to see him again and found that he had been operated upon, and, because of the extent of the lesion, a colostomy was the only thing that could be done.

I cite this experience to emphasize the necessity of being most suspicious of the presence of malignancy when there is bleeding from the bowel, even though the clinical picture is that of an inflammatory process.

DR. ANDERSON, closing: The only thing I would like to add is that I had two slides made to use in this discussion. I appreciate the discussions of the men this evening.

These slides show the common and less common lesions producing rectal bleeding.

Lesion Producing Rectal Bleeding

Less common:
- Submucous lipoma
- Submucous fibroma
- Leukemia
- Purpura
- Intussusception
- Foreign body
- Typhoid fever
- Malaria
- Hepatic disease
- Drugs
- Parasites
- Endometriosis
- Factitial proctitis

Common:
- Internal hemorrhoids
- Anal fissure
- Anal fistula
- Rectal fistula
- Rectal adenomata
- Carcinoma—rectum and colon
- Colitis—
  - Ulcerative, idiopathic
  - Amebic
  - Bacillary
- Proctitis
- Diverticulosis
- Prolapse
- Trauma
- Postoperative

VIRUS ASPECTS OF CARCINOMA

ROBERT G. GREEN, M.D.
University of Minnesota
Minneapolis, Minnesota

Cancer research from a virus viewpoint has as yet attracted the efforts of relatively few investigators, but an increasing interest in the possible virus causation of carcinoma, as well as of tumors in general, has been apparent during the past ten years. Viruses have now been shown to be intimately associated with the tumor-cancer problem, and the consistent presence of viruses in certain malignant growths must finally be resolved to a role of causation or definitely relegated to a position of simple association. Comparatively little effort has been expended on the direct investigation of human cancer from the virus viewpoint or from other basic approaches. Physiological, virological and immunological studies on human cancer are very limited. Most investigations in the field of cancer research are conducted on tumors of animals and birds.

Viruses are now becoming one of the more important aspects of cancer research. From the information at present available on such malignant tumors as the carcinoma of rabbits that originates from the Shope

papilloma and the breast carcinoma of mice that is stimulated by the presence of the Bittner virus, the filterable viruses must at least be considered biological carcinogenic agents along with coal tar and certain refined chemical compounds. Viruses have a broad distribution among animals and human beings, and some species seem to lurk for long periods or even a lifetime within tissues without producing apparent symptoms. The distribution of viruses and their tumor-producing potentialities point out the viruses as a possible factor in the ultimate causation of most malignant tumors. If, however, viruses should be the basic cause of all or some cancers, the problem still is not a simple one. Hereditary susceptibility, hormonal influences and nutrition all seem to play an important role in the final appearance of the malignant cell.

Many benign and malignant tumors of animals and birds have now been described that have an associated virus and can be reproduced by injection of the virus into the specific animal host. A virologist does not hesitate to accept a virus causation for such tumors as the rabbit papilloma, the oral papilloma of dogs, or the Rous tumor of chickens. However, the ease with which cancer can be produced by the application of chemical agents, and the apparent absence of viruses in cancer thus artificially produced reacts to discredit a theory of virus causation. On the other hand, it cannot be said that the presence of a virus in any tumor has at this time been absolutely disproved. Failure to transmit a tumor upon limited trial under specific conditions does not necessarily mean that an infective agent is not present. Such a situation exists in the case of the cottontail papilloma established in domestic rabbits as described below. In virus tumors we are dealing with host-parasite relationships of considerable complexity, with most of the operative factors yet unknown. Nevertheless, at this time viruses seem to some investigators the most tangible factor as a general cause of both benign and malignant growths.

Researches on tumors are more than ever bringing up questions as to the nature of viruses. There has been a great tendency among those in the field of cancer research to regard a virus as a cell reproductive component gone wild. Such might be termed a feral gene. As yet, however, there is no significant evidence that viruses have their origin in host cells. Evidence presented by the data of this paper tends to establish that the infective agent of mammary carcinoma in mice is a parasite entirely foreign to normal mouse tissue. The origin of viruses from microbes and their nature as highly adapted parasites are reasonable concepts and correspond to the course of parasitism and the nature of highly adapted parasites as observed throughout the biological world.

Many tumors, both benign and malignant, that occur in various species of lower animals, birds and mammals, have been shown to be due to the presence of a filterable virus. The viruses causing the tumors generally are very specific and will reproduce the tumors only if inoculated into one species of animal and into one kind of tissue. The oral papilloma of dogs, for example, appears very specific for the dog and has not been successfully inoculated into other species nor into any tissue except oral epithelium. The papilloma of cottontail rabbits can be easily transmitted in series through cottontail rabbits. It can be transferred to domestic rabbits for one generation but cannot easily be maintained in them by serial inoculation. When the papilloma is inoculated from the cottontail rabbit into the domestic rabbit it often becomes cancerous. The papilloma virus cannot be isolated from the cancer, but cancer transplants can be transmitted in series in domestic rabbits by the use of living cancer cells as the inoculum. As the cancer transplant grows it appears to immunize the rabbit against the virus of the papilloma.[7]

Mammary cancer of mice has received a great deal of experimental study, and during the past five years has been the principal object of our investigations on virus tumors. Susceptibility to spontaneous mammary cancer in mice is a strong hereditary factor, and high- and low-cancer strains of mice have been developed by selective breeding. In a high-cancer strain, as many as 95 per cent of female mice may develop breast cancer after producing three or more litters. The development of this cancer of mice is greatly affected by hormones and nutritional factors. The cancer occurs in mice, however, only if a specific agent is present, as was first demonstrated by Bittner.[2] The agent is regularly transmitted from cancerous mothers to young mice through the milk and has been variously referred to as the milk influence, the milk factor, or the milk agent. Bittner[3,4] showed that the agent was filterable, and our co-operative studies[5] have demonstrated that the agent is of the magnitude of a virus. Injected into young mice of a susceptible strain, the agent will stimulate cancer-production after a period of seven months to a year. It is of the greatest importance that the agent has been found to be antigenic. Andervont and Bryan[1] prepared two antiserums from two rabbits and found that these serums neutralized the milk agent. In the antiserum groups he obtained only one tumor in thirty-six mice injected with a mixture of antiserum and milk agent, and ten in fifteen mice injected with the agent alone. We have extended this work with more controls and have established beyond question that the agent is antigenic. Upon injection of the agent into rabbits or rats, tumors do not develop but these animals are stimulated to produce antibodies. Our serums were prepared by immunizing groups of three or more rabbits by multiple injections of high-speed centrifugates of mouse tumor tissue. The centrifugal speeds were such as would concentrate particles of virus size. Antiserum from the immunized rats or rabbits neutralized the milk agent completely. The milk agent, therefore, has all of the cardinal characters of a filterable virus and may correctly be referred to as a tumor-producing virus.

Recently we have found that the mouse cancer antiserums not only neutralize the virus of mammary carcinoma[6] of mice but also inactivate the cancer cells[8] when they are suspended in the antiserum. Cancer cells are not inactivated by treatment with normal rabbit serum nor by treatment with serum from rabbits and rats immunized against normal mouse tissue. The cytotoxic property of the mouse cancer antiserum for cancer

cells has been demonstrated in several experiments of which the following is an example.

A cancer cell suspension is prepared from masses of cancerous tissue removed sterilely from mice. The cancer tissue is forced through a tissue press. The macerated tissue is suspended in 0.9 per cent saline solution and placed in long test tubes to settle. After thirty minutes, the larger masses of cancer cells will have settled to the bottom and above will be a suspension of free tumor cells and small aggregates of tumor cells. If a susceptible mouse is injected with a small dose of these cells, a cancer will grow as a transplant tumor. In this experiment, ten mice were thus injected as a control group to assure the activity of untreated cells. For the actual test of cytotoxic activity of the serum, a portion of cancer cell suspension was mixed with five times the volume of mouse cancer antiserum and held for three hours at room temperature and for three hours at refrigerator temperature. Ten mice were then injected with 0.1 c.c. of the mixture. Two additional controls were set up by treating portions of the cancer cell suspension in a similar manner with normal rabbit serum and with serum from rabbits immunized with normal mouse tissue. The results are shown in Table I.

TABLE I. CYTOTOXIC PROPERTY OF MOUSE CANCER ANTISERUM

Rabbit antiserum—C3H tumor—ZBC mice without milk factor

No. mice	Materials injected	No. tumors 10 days	No. tumors 15 days	No. tumors 40 days	No. tumors 60 days
10	Tumor cell suspension + cancer antiserum	0	0	0	0
8*	Tumor cell suspension + normal mammary antiserum	1	3	8	8
10	Tumor cell suspension + normal rabbit serum	1	1	5	7
10	Tumor cell suspension + saline solution	10	10	10	10

*2 died from intercurrent infection.

From these results it is seen that the cancer antiserum prevented completely any tumorous growth of the cancer cells. The cancer cells suspended in saline solution grew rapidly to produce transplant cancers in all ten control mice. Most of the mice injected with cancer cells in normal mammary tissue antiserum and normal rabbit serum developed transplant cancers, but the cancerous growths appeared definitely later and were fewer in number. In this and other experiments it has been a consistent finding that normal rabbit and normal rat serums and the serums of rabbits immunized with normal tissues contain natural antibodies, or something of a similar nature, that affect cancer cells adversely and retard their growth to a limited degree. This effect, as found in normal tissue antiserum, is removed by absorption with normal mouse breast tissue cells but not with cancer cells. Thus, the slowing effect of normal tissue antiserum seems due to a nonspecific factor that combines better with normal cells than with cancer cells.

Absorption experiments with the mouse cancer antiserum reveal that the antibodies present are absorbed by cancer cells but not by normal breast tissue cells. This seems to establish the presence of antibodies that are specific for cancer cells and will not attack normal cells. The implication is that mouse cancer cells have taken on a new specificity and are immunologically different from normal mouse cells. It seems of considerable significance that our findings indicate that mouse cancer cells are antigenically different from normal cells and that the same antiserum will neutralize a virus and inactivate the cancer cells stimulated by that virus.

Originally thought to be more or less a character of protoplasmic disorganization transmitted genetically, the mouse cancer now clearly seems to be a virus infection with susceptibility and resistance strong genetic factors. With the addition of these findings on mammary carcinoma of mice to those previously established for other virus tumors, it would seem that we must more and more turn our eyes toward virus investigations in cancer research.

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Discussion

DR. MAURICE B. VISSCHER, U. of M.: I am very much impressed by the newer work Dr. Green has been doing. It is now quite apparent that it is possible to produce antibodies against the milk influence. The part of Dr. Green's work that I had not known about until this evening is the demonstration that this antibody is effective not only in preventing the action of this virus-like agent in producing the carcinoma, but is also active in preventing the growth of carcinoma cells in cell suspensions. The two processes are quite different because in one case a carcinogenic agent is inhibited and in the other a complete cell is prevented from growing. To show that the actual carcinoma cell can be prevented from further growth by the action of an antibody of the sort produced in another species is really a most important step forward. I wouldn't suggest that one would necessarily find that this would have any bearing on the stopping of the growth of masses of cells but it is a step in that direction. I wonder if Dr. Green has anything to say about what masses of cells will do under comparable circumstances. He has given us an excellent presentation of very important new work.

The meeting adjourned.

J. A. LEPAK, *Secretary*

WOMAN'S AUXILIARY

MRS. EDWARD V. GOLTZ, *President*
Saint Paul, Minnesota
MRS. JOHN K. BUTLER, *Editor*
Carlton, Minnesota

STATE BOARD

On January 30, Mrs. E. V. Goltz, president, presided at the winter meeting of the State Board of the Woman's Auxiliary of the State Medical Association, which was held at the Radisson Hotel at the University of Minnesota in Minneapolis. The guest speaker at the luncheon was Dr. Myron M. Weaver, Assistant Dean of Medicine, who outlined the latest developments of the Murray-Wagner-Dingell Bill and its implications. His talk was intensely interesting and informative.

BLUE EARTH COUNTY

The Blue Earth Auxiliary met at the Mankato Clinic on January 28. This was a business meeting only, and was devoted to a discussion of public relations and the cancer drive. Mrs. A. F. Kemp is county chairman of Cancer Control. On January 21, a joint meeting of the Blue Earth and Nicolett-Le Sueur program and executive committees was held at a luncheon at the Elks Cafe, Mankato. At this luncheon, a date was set for a public relations meeting at which Dr. Haddow M. Keith of the Mayo Clinic will be the speaker.

HENNEPIN COUNTY

On January 4 the Hennepin County Medical Auxiliary held a luncheon meeting at the Radisson Hotel. Mrs. Donald Bacon, wife of a Saint Paul physician, read the play *Dear Ruth.*

Mrs. N. H. Lufkin was chairman for the lunch and Mrs. Martin Nordland had charge of hospitality.

Mrs. Harold F. Wahlquist, a former president of Heneepin County Auxiliary, now national treasurer of the board of directors of the Women's Auxiliary of the American Medical Association, attended the mid-year meeting in Chicago.

The death of Dr. J. F. Curtin, husband of the president of the Auxiliary, saddened the members.

RAMSEY COUNTY

Miss Ruth Freeman, assistant professor of Public Health Nursing at the University of Minnesota, addressed the Ramsey County Auxiliary at the Ramsey County Library, January 28. Her subject was "New Developments in Public Health."

Mary Ellen Hoaglund, winner of the Junior award, and Beverly Hanklin, winner of the Senior award, of Saint Paul, read their essays on tuberculosis and were presented with war stamps.

ST. LOUIS COUNTY

On January 8, the St. Louis County Auxiliary met at the home of Mrs. Martin A. Wallace, Duluth. After the business meeting "Juvenile Delinquency" was the subject for discussion. On Saturday evening, January 26, Auxiliary members entertained their husbands at the annual dinner dance at the Kichi Gammi Club.

MOWER COUNTY

Mrs. L. G. Flannagan, of Austin, was hostess to her Auxiliary on January 28. Mrs. C. L. Sheedy of Austin gave the Program on Plans for Medical Care taken from *Hygeia.*

The Auxiliary, of which Mrs. C. C. Allen of Austin is president, is also active in its work on cancer dressings.

STEARNS-BENTON

Mrs. J. B. Gaida, president, was hostess to the Auxiliary at the Hays Hostess House on January 8. Miss Kay McEnroe, flight instructor of St. Cloud Airport, introduced by Mrs. H. B. Clark, talked on "What is New at St. Cloud Airport."

Stearns-Benton has lost two of its members. Mrs. C. S. Sutton, a former president, died January 7. Mrs. J. B. Beuing died January 28. Mrs. Henery Korda was introduced by Mrs. Carl Luckenmeyer, membership chairman as a new member. Mrs. Charles Donaldson and Mrs. Hayward Kaliher, Foley, were the out-of-town members present.

WASHINGTON COUNTY

Mrs. F. M. McCarten, Stillwater, was hostess to the Washington County Auxiliary on January 8. Mrs. R. Samson joined the Auxiliary as a new member.

WINONA COUNTY

The Winona Auxiliary met for dinner at Hotel Winona on January 7 and then adjourned to the home of Mrs. J. T. Benoit for the business meeting.

ACUTE ARTERIAL OCCLUSION

(Continued from Page 252)

Embolectomy and thrombectomy were not performed in any of these cases, and in only one instance was local anesthetization of the sympathetic ganglia necessary.

This is a small series of cases when compared with the series of McKechnie and Allen—but the results would indicate that use of the plan of treatment for acute arterial occlusion which we have recommended will result in saving some extremities and prolonging some lives. Except for the addition of anticoagulant therapy this plan of treatment is similar to that previously used on the vascular service for many years.

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In Memoriam

JOHN MILTON ARMSTRONG

In the passing on December 15, 1945, of John Milton Armstrong, the medical profession of Saint Paul lost a colorful member who had devoted much of his time and talent to the interests of the profession.

Born in Saint Paul, April 10, 1875, the son of George W. and Jane C. Armstrong, who came to Saint Paul in 1853, Dr. Armstrong graduated from Central High School in 1894. He obtained his medical degree from the University of Minnesota in 1901 where he was a member of Chi Psi and Nu Sigma Nu fraternities. After serving an internship at St. Joseph's Hospital in Saint Paul in 1901-2, he practiced for three months in Montgomery, Minnesota.

In 1902 he began practicing in Saint Paul and from 1907 to 1911 served as Assistant Commissioner of Health. Early interested in Dermatology and Genito-Urinary diseases, he was instructor in this department in the University of Minnesota medical school from 1903 to 1916. In 1913-14 he took postgraduate work in his specialty in Vienna, returning just prior to World War I. He was a fine diagnostician in his specialty.

Dr. Armstrong was a member of the Minnesota Academy of Medicine of which he was president in 1941, the Minnesota Dermatological Society, the Ramsey County Medical Society of which he was president in 1914, the Minnesota State and American medical associations.

He was greatly interested in medical literature and did more for the Ramsey County Medical Library to raise it to its present enviable status than any other individual. It was he who began and largely developed the library museum and obtained endowments in memory of former members for the purchase of rare volumes. He had served as a member of the library committee since 1906 and maintained his interest in the library even after his retirement from active practice in 1941. He worked particularly hard on the completion of files of old American medical periodicals and it is this collection especially that makes the library such an outstanding one. He contributed to the library many of his own books and many interesting items pertaining to very early Minnesota physicians, especially Saint Paul physicians.

Dr. Armstrong was business manager of the *Saint Paul Medical Journal* from 1908 until 1912 and was member of the Editing and Publishing Committee of MINNESOTA MEDICINE from 1924 until 1933. As a member of the original Historical Committee of the Minnesota State Medical Association appointed in 1928, he began the publication in 1938 of the History of Medicine in Minnesota in MINNESOTA MEDICINE and stimulated others to continue the work.

In January, 1932, he was elected to the Board of Trustees of the Hill Reference Library; in May, 1942, he was asked to serve as chairman of the library committee, and in 1943 he was elected chairman of this

committee. Only the most severe illness kept him from fulfilling his committee duties and he attended the meetings until a few days before his death. Miss Starr, Librarian of the Hill library, calls him "a true bookman" and she says further: a "brilliant man, endowed as few are, he was modest. His suggestions were most helpful in building up the natural history collection of the library."

He was a charter member of the medico historical club *Kos Knidos,* and remained very active in this association which flourished in the Twin Cities until the beginning of World War II.

He was deeply interested in all branches of natural science which included every phase of natural history, astronomy and the microscopic study of fungi and parasites. He ground and constructed a very creditable reflecting telescope with which he and his friends studied the heavens. He was interested in the geological structure of the earth and had collected many specimens of fossils from the Ordovicion about Saint Paul. He possessed a beautiful collection of polished agates obtained from the local glacial drift. His interests led him into the study of ancient and modern archery and he accumulated an important, representative collection which he eventually presented to the Hill Reference Library. This gift occasioned an exhibition on Archery in this library. It was described by James Gray *(Saint Paul Dispatch,* February 19, 1943) as containing old, rare volumes (going back as far as the early 17th century), as well as scholarly, literary and technical items. James Gray labeled Dr. Armstrong "an unobtrusive, genial and humorous gentleman who has always loved the out-of-door life." His home-made bows and arrows made from various woods of North and South America were beautifully made. His intellectual curiosity led him to read and familiarize himself with the Einstein Theory of Relativity, when it first came out.

Books were his consuming interest. He was an omnivorous reader, with interest in all literature dealing with American medical history. In his search for material for the History of Medicine in Minnesota he literally went through hundreds of volumes of older American medical periodicals. His historical articles on the "History of Cholera in America" and "Dr. Purcell, the first Minnesota Physician," were very well received. His very interesting article on Dr. W. S. Cox (MINNESOTA MEDICINE, January, 1945) is proof of his gift of ferretting out information from every imaginable source. His wide reading in medical history is shown in one of his very early publications, "The Genesis of the Transmissible Disease" (1909), the prize essay of the Ramsey County Medical Society for 1908, which also shows his earnest application to his work in the Saint Paul Department of Health and his interest in bacteriology.

One of his finest qualities lay in his ability to stimulate similar interests in others. He enjoyed revealing the results of his searches. Being a storehouse of local, especially local medico-historical information, he was always ready to share it with those looking for it. With his varied hobbies he was a most colorful personality to all his friends and those who knew him well.

Dr. Armstrong married Ida Dobyns, of Shelbyville,

Missouri, on May 31, 1909. He is survived by his widow and four children, Jane, Anne (Mrs. W. A. Wallis), Elizabeth (Mrs. Russell T. Nichols) and John Milton, Jr.

WILLIAM JAMES COCHRANE

Dr. William J. Cochrane, for many years a resident of Lake City, passed away February 1, 1946, at the Lake City Hospital.

Dr. Cochrane was born in Lake City, Minnesota, June 27, 1867. He attended high school in Winona and received his M.D. from the College of Physicians and Surgeons in Chicago in 1895. He served as intern for eighteen months in the Illinois Eye and Ear Infirmary in Chicago, and then located at Quincy, Illinois, in 1896. He moved to Lake City in 1899.

He served as Captain in the Army Medical Corps during World War I and in 1920 and 1921 practiced in Iowa, returning to Lake City in 1921.

Dr. Cochrane was a member and past president of the Wabasha County Medical Society, and a member of the Minnesota State and American Medical Associations. He had been chairman of the Sanatorium Commission of Winona and Wabasha Counties since 1927, and had been president of the Lake City Hospital board. He also was local surgeon for the Chicago, Milwaukee, St. Paul & Pacific Railway. A Mason for fifty years, he was a Past Master of the local lodge.

In 1916 he took postgraduate work in eye, ear, nose and throat at the Illinois Post Graduate Hospital in Chicago.

Dr. Cochrane married Nellie A. Phelps of Lake City in 1901. He is survived by his widow and two daughters, Mrs. Leo F. McCaffrey of Brainerd and Mrs. John R. Wala of Beverly Hills, California, and one son, William, of Chicago.

Dr. Cochrane had been a member of the Congregational Church in Lake City since 1901 and had served as deacon and trustee. In 1943 he was elected president of the Lake City Bank and Trust Company. He was active in civic affairs and held the respect and admiration of all who knew him.

THE Rh FACTOR

(Continued from Page 249)

tor and paid very little attention to the changes in the brain.

It becomes perfectly obvious that any blood bank or clinical laboratory which maintains a list of blood donors should include in that list the names of Rh negative individuals who may volunteer as donors. Such donors should preferably be male, and if female, should not have been pregnant, unless the husband happens to be Rh negative. Prompt transfusion with Rh negative compatible blood may save a life, either a hemorrhaging patient or an erythroblastic baby.

* * *

In the preparation of this paper, the senior author is deeply indebted to Dr. Isreal Davidsohn, pathologist of Mt. Sinai Hospital, Chicago, whom he freely consulted for suggestion and criticism.

Of General Interest

Dr. Paul C. Benton has re-opened his offices at Gibbon, after an absence of three and a half years in the Army Medical Corps.

* * *

Dr. Henry Roemer has returned home to Winona from thirty-nine months of service in the Navy and has resumed his practice at the Winona Clinic.

* * *

After three years in service as a flight surgeon, Dr. Lawrence Larson has resumed his practice in Minneapolis, with offices in the Medical Arts Building.

* * *

Dr. John J. Ederer, formerly of Mahnomen, Minnesota, has retired from practice because of ill health and is now residing in Minneapolis.

* * *

Dr. Russell O. Sather has assumed the duties of health officer at Crookston, replacing Dr. Lyle Brown, who is ill.

* * *

Dr. Robert Fuller Mears, Northfield, has purchased a site for the erection of a modern clinic building as soon as materials are available.

* * *

Dr. David H. Rolig has closed his offices at Howard Lake and moved with his family to Saint Paul, where he is associated in practice with Dr. Olaf Sohlberg.

* * *

Dr. Joseph F. Bicek, 1267 Lowry Medical Arts Building, Saint Paul, announces the limitation of his practice to obstetrics and gynecology.

* * *

Dr. J. Arnold Malmstrom and Dr. Edward N. Peterson of Virginia, attended the sectional meeting of the American College of Surgeons held in Minneapolis during the last week of January.

* * *

Dr. Robert P. Buckley was elected president of the Board of Directors of the Duluth Mental Hygiene Clinic at the annual meeting. Dr. Buckley was formerly associated with the hospital in Coleraine.

* * *

Drs. Raymond J. Josewski and Lloyd Taylor, Stillwater, represent the medical profession on the health committee for Washington County, which has been organized by the County Board of Commissioners.

* * *

Dr. Thomas Byrd Magath, of the Mayo Clinic, was elected president of the State Board of Health at the annual meeting of the Board held in Minneapolis on January 24.

* * *

Discharged from the Navy after thirty-five months of service as lieutenant commander, Dr. Harold M. Skaug has returned to Chatfield and has resumed his medical practice.

* * *

Dr. Oscar F. Mellby, president of the Oakland Park Sanatorium at Thief River Falls, was re-elected at the annual business meeting. Dr. Baldwin Borreson, superintendent of the sanatorium, is secretary ex officio.

* * *

Dr. William R. Jones was re-elected chief of staff of Eitel Hospital at the annual staff dinner held at the Minneapolis Club. Dr. Frank Hirschfield was made assistant chief of staff, and Dr. Harold Reif secretary.

* * *

Effective February 1, Dr. Howard G. Bosland terminated his practice at Verndale to become associated with Dr. Berton J. Branton at his clinic in Willmar. Dr. Bosland had been practicing in Verndale since October, 1932.

* * *

Dr. Anthony H. Field, who for the past year was stationed in England with the 109th General Hospital, has resumed his practice of medicine and surgery at Farmington. His offices are in the Sanford Hospital Building.

* * *

Dr. Clarence W. Walter, Saint Paul, has resumed practice at 1210 Lowry Medical Arts Building after serving as Lieutenant Commander in the Navy. He was stationed for two years in the Aleutian Islands and then at the Navy Hospital at Long Beach, California.

* * *

Dr. Emmet V. Kenefick has re-opened his offices for the practice of internal medicine at 710 Lowry Medical Arts Building, Saint Paul. A captain in the Air Force, Dr. Kenefick was stationed at Tinker Field, Oklahoma City, and at the Santa Ana Air Base in California.

* * *

Dr. John R. Kelly, who before his induction into the Army Medical Corps was practicing at Aitkin, Minnesota, has opened offices at Cold Spring. Dr. Kelly was in service for three and a half years with eighteen months of foreign duty. During this time he was stationed in Canada and Alaska.

* * *

Dr. Gaylord Anderson, former Director of Public Health at the University of Minnesota, was guest speaker at the midwinter meeting of the Minnesota Organization for Public Health Nursing held in Coffman Union. Dr. Anderson only recently returned from military service.

* * *

Colleagues and friends of Dr. L. J. Alger, of Grand Forks, North Dakota, will be interested in knowing that

(Continued on Page 288)

LFI MODEL SW-221 SHORT-WAVE DIATHERMY

Many good things have been unobtainable all during the war and are just now becoming again available. Here's one of them—an unquestionable "find" for the physician or hospital in need of diathermy equipment is the Model SW-221 with

THE HINGED TREATMENT DRUM

The Model SW-221 is a powerful, compact, easily controlled apparatus. It comes equipped with the handiest, most adaptable applicator known to diathermy—The Hinged Treatment Drum (exclusive with Liebel-Flarsheim).

Note These Convenient, Effective Applications:

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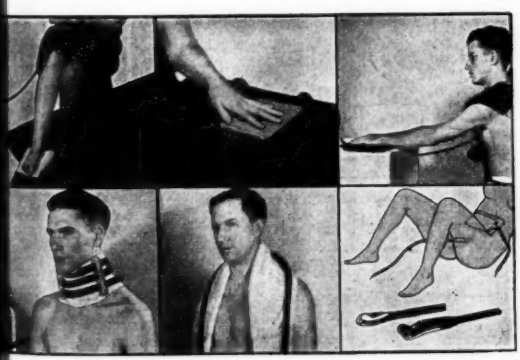

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Condenser pads, cuffs and inductance cable are standard equipment. Orificial applicators and accessories for minor electrosurgery are available optionally.

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(Continued from Page 286)

Dr. Alger, on a recent trip to California, where he attended the Clinical Postgraduate Convention in Los Angeles, solved transportation difficulties by flying his own Stinson Voyager.

* * *

Dr. L. G. Culver, Saint Paul, has resumed practice in association with Dr. Carl L. Larsen and Dr. Merritt W. Wheeler, specializing in ophthalmopathy and Otolarynogology, following discharge from the Navy where he served aboard the *Ticonderoga* and the *Kenton* as Commander.

* * *

Dr. Balcome, son of the late Dr. F. E. Balcome, who practiced in Saint Paul for thirty years, served in the South Pacific. He is a graduate of Macalester College and the University of Minnesota Medical School, and had been practicing in Saint Paul for nine years prior to his enlistment.

* * *

Dr. Donald Dukelow, director of public health education in the Minnesota Department of Health for the past eight and a half years, has been appointed secretary of the health and medical care division of the Minneapolis Council of Social Agencies. Dr. Dukelow is also a member of Mayor Humphrey's health advisory committee which was recently organized in an effort to combat the sharp increase in communicable diseases in Minneapolis.

After an absence of more than three years in the military service, Dr. Manford B. Dahle has returned to Glenwood and re-opened his offices in the Glenwood Theater Building. Following a year in the South Pacific, Dr. Dahle was on duty for the remainder of his enlistment in this country.

* * *

Dr. Harvey O. Beek has resumed the practice of internal medicine in his offices in the Lowry Medical Arts Building, Saint Paul Dr. Beek entered service as a member of the University Hospital Unit No. 26, but later was made chief of medical service at the 45th General Hospital located in Bari, Italy, with rank of lieutenant colonel.

* * *

Dr. Albrecht E. Muller, who has been practicing in Saint Paul since his discharge from five years in military service, has purchased a home in North Saint Paul and will open offices there as soon as equipment can be obtained. Dr. Muller is a graduate of the University of Minnesota Medical School and interned at St. John's Hospital in Saint Paul.

* * *

Dr. Joseph P. Spano, who was discharged from military service last November, has returned to his practice in Minneapolis and has re-opened his offices at 328 East Hennepin Avenue. Dr. Spano went overseas with the 250th Staff Hospital in November, 1943, and served in

England, France, Belgium, Germany, Bavaria and Austria. He entered service in June, 1942, and was stationed at Fort Leavenworth for the first year.

* * *

The chief topic of discussion at the Health Workshop, which was sponsored by farm and labor groups at their joint convention in Saint Paul during the first week of February, was a medical program suitable for the masses and its cost. Delegates from Wisconsin, Minnesota, and both Dakotas united in a demand for political action which would initiate improvement of health standards.

* * *

Dr. A. C. Hilding was elected chief of staff of St. Luke's Hospital in Duluth, at the annual dinner meeting of the medical staff. The other officers elected at this time were Dr. Arnold O. Swenson, vice chief of staff; Dr. Charles Miller Bagley, secretary; Dr. Frank J. Hirschboeck, chief of medicine; Dr. Gordon C. MacRae, chief of obstetrics; and Dr. Arden L. Abraham, chief of x-ray.

* * *

Dr. Lawren R. Nesset, who was released from military service in January of this year, is now associated in the practice of medicine and surgery with Dr. Burton C. Ford at the Marshall Clinic in Marshall, Minnesota.

Dr. Nesset, who comes from Canby, Minnesota, graduated from Luther College in Decorah, Iowa, and the University of Minnesota. He interned at General Hospital in Minneapolis, where he was also in residence for a year. Overseas duty took Dr. Lawren into Germany and Czechoslovakia.

* * *

Two members of the Mayo Clinic, Dr. Charles W. Mayo and Dr. Donald C. Balfour, have been signally honored by the Chilean government which has made them commanders in the *Order de Merito*. The presentation of the order—the highest attainable by the medical profession in Chile—was made by Dr. Alejandro Garreton, professor of internal medicine at the University of Chile in Santiago, while he was in Rochester to observe methods of postgraduate study and procedures in practice of internal medicine.

* * *

Dr. Frederick P. Arny and Dr. Milton M. Balcome, recently discharged from duty as flight surgeons in the Army, are now associated in practice in St. Anthony Park, with offices at 2315 Como Avenue.

Dr. Arny, who graduated from the University of Minnesota Medical School in 1935, entered practice at Princeton, Minnesota, where he remained until his induction into the Army seven years later. He is the son of Professor Arny, a member of the faculty of the University. Dr. Arny's area of duty was mainly confined to the ETO.

* * *

One of the first British physicians to take advantage of the resumption of the reciprocal fellowships between England and the University of Minnesota, which were discontinued during the war, is Dr. Richard Mayon-White, of Sussex, England, now studying in the Department of Pediatrics. A pioneer in this branch

of medicine in England, study in this country is essential for him, since children's diseases are not treated as a special branch of medicine in England. Dr. Mayon-White and five other English physicians were awarded fellowships by the Rockefeller Foundation.

* * *

Four members of the Mayo Clinic, Dr. Wallace E. Herrell, Dr. Charles W. Mayo, Dr. Donald C. Balfour and Dr. Waltman Walters, took part in the program at the sectional meeting of the American College of Surgeons held in Minneapolis in January.

Dr. Herrell discussed "Treatment of Surgical Infections by Chemical and Antibiotic Agents." Dr. Walters was a discussion leader on "Treatment of Open Wounds." Dr. Mayo took part in a panel discussion on pre- and postoperative supportive treatment, and Dr. Balfour was the moderator of a discussion and open forum on graduate training in surgery.

* * *

The annual George Chase Christian Lecture in the field of cancer research was delivered at the University of Minnesota by Dr. Leonell C. Strong, associate professor of anatomy, Yale University School of Medicine, on February 7, 1946. Dr. Strong's subject was "Mice, Men and Malignancy."

A pioneer in the use of inbred strains of mice for cancer research, Dr. Strong's first contribution to the study was the theory that failure in growth of grafts of tumor tissue is a consequence of inherited differences between the tissues of the host and the tumor cell, and depends on genetic differences in tissues.

* * *

The following appointments to the consultant staff of the Veteran's Hospital in Minneapolis have been approved by the Veterans Administration: Dr. Charles D. Creevy, senior consultant in urology; Dr. Thomas Kinsella, senior consultant in thoracic surgery; Dr. Carl W. Waldron, senior consultant in plastic surgery; Dr. Nathaniel H. Lufkin, senior consultant in pathology; Dr. Joseph W. Baird and Dr. Arthur W. Friend, consultants in anethesiology; Dr. William J. Norman, consultant in urology, and Dr. William Hollingshead, consultant in medicine; all of Minneapolis. Dr. Rodney Kendall, of Saint Paul, has been made consultant in dermatology.

* * *

Following the completion of his terminal leave on February 1, Major Thomas R. Dolan entered medical practice at Caledonia in association with Dr. Jacob J. Ahlfs.

Dr. Dolan, a former resident of Green, Iowa, is a graduate of Loras College, Dubuque, and the St. Louis University Medical School, St. Louis, Missouri. Beginning his internship in 1940, he was at the Charity Hospital in New Orleans for a year and a half. Of four and a half years in military service, Dr. Dolan spent thirty months as flight surgeon with the 13th Army Air Force Heavy Bombardment Squadron in the Pacific Theatre. During the latter part of his service, Dr. Dolan was engaged in research in aviation medical problems.

Dr. Hugh Monahan has returned to practice in association with his father, Dr. Robert H. Monahan, and Dr. Claire C. Craig at International Falls, after two and a half years in the Army Medical Corps.

Dr. Monahan, who was a member of the medical reserve, was called into active service in July, 1943. He was in residence for fifteen months at Fitzsimons General Hospital in Denver, as assistant chief of the eye, ear, nose and throat section. Later he went overseas with the 70th Infantry Division as battalion surgeon and was stationed in France and Germany. When the war ended he was assigned to the 280th Station Hospital in Germany and the 15th General Hospital at Udine, Italy. Dr. Monahan intends specializing in diseases of the eye, ear, nose and throat.

* * *

Announcement has been made of the election of Dr. Jay Arthur Myers, Minneapolis, as chairman of the editorial board and editor-in-chief of *Diseases of the Chest*, as successor to the late Dr. Ralph C. Matson.

Other members of the editorial board are: Dr. Andrew L. Banyai, Wauwatosa, Wisconsin; Dr. Charles M. Hendricks, El Paso, Texas; Dr. Champ H. Holmes, Atlanta, Georgia (inactive), and Dr. Richard Overholt, Brookline, Massachusetts.

Scientific manuscripts should be sent to Dr. Jay Arthur Myers, 111 Millard Hall, University of Minnesota, Minneapolis 14, Minnesota. All notices and miscellaneous items for publication in the journal should be sent to the Executive Offices, American College of Chest Surgeons, 500 North Dearborn Street, Chicago 10, Illinois.

* * *

Senior members of the Academy of Medicine and seven honorary members were guests of honor at the dinner meeting of the Academy held at the Minneapolis Club on January 23. The honorary members, all from the University of Minnesota, are Elexious T. Bell, professor of pathology; Harold S. Diehl, dean of medicine; Henry L. Ulrich, clinical dean of medicine; Benjamin J. Clawson, professor of pathology; George D. Head, clinical professor of medicine; and Richard E. Scammon, dean of medical science. The seventh member, Adolph M. Hanson, Faribault, associate professor of clinical medicine, was unable to attend, because of illness. Lieutenant Colonel Edward Evans, associate professor at the University of Minnesota Medical School and senior consultant in orthopedic surgery at the Veteran's Hospital, Minneapolis, was chairman of the meeting. Dr. Evans had just returned from four years' service in the Army.

* * *

Dr. Robert D. Estrem and Dr. J. D. Mouritsen, recently discharged from military service, have rejoined the Estrem Clinic at Fergus Falls.

Dr. Estrem, a captain in the Army Medical Corps, served sixteen of his twenty-two months in service overseas. He was in general surgery at the 90th General Hospital in England his first six months across. Then he was assigned to the Sixth Armored Division under General Patton at the beginning of the Battle of the Bulge. The following five months he was in front-line service

in a forward-aid station with the 86th Cavalry Division on the march through France, Belgium, Luxembourg and Germany. He was awarded the Bronze Star for particularly meritorious service.

Dr. Mouritsen entered the Navy in September, 1942, and was discharged in December, 1945. A lieutenant commander, he served aboard the USS *Colorado* in the Pacific for twenty-six months. He was in action at Tarawa, Kwajalein, Eniwetok, Saipan, Guam, Tinian, Leyte, Mindoro, Luzon and Okinawa, and was in Tokyo Bay when the peace terms were signed.

* * *

Friends and neighbors—five hundred of them—disregarding a temperature of fifteen degrees below zero, gathered from miles around for a party held in the high school at Willow River in honor of Dr. William C. Ehmkes's sixty-eighth birthday. Features of the program were addresses by Dr. Thomas Moe, of Moose Lake, and Dr. Paul G. Boman, of the Duluth Clinic. A biographical sketch of Dr. Ehmke's life was read, and the doctor was presented with a purse of money in appreciation of his many years of devoted service.

Dr. Ehmke was born in Le Sueur County. He graduated from Hamline University and took his medical degree at the University of Minnesota. Following his internship, he was made company doctor for the Atwood Lumber Company's camps in Pine County. That was more than forty years ago. Finding the country and the people to his liking, he established a practice and has continued it ever since. The "good doc," as he is affectionately known throughout the community, has not only ministered to the health needs of its people, but has also taken an active interest in civic affairs.

* * *

The Hennepin County Medical Society has announced the return from service of the following members:

Dr. Lyman B. Clay has resumed his practice at 2703 East Lake Street. Dr. Clay was a lieutenant commander in the Navy with duty in the South Pacific.

Dr. Harold F. Buchstein, neurological surgeon, had served in naval hospitals at Great Lakes, Illinois, Corona, California, in the South Pacific and on Okinawa, since January, 1942. His offices are in the Medical Arts Building.

Dr. Meyer C. Goldner, orthopedist, has returned to his offices in the Medical Arts Building. He was a staff member of General Hospital 26 in North Africa, and was chief of orthopedics with the group for some time.

Dr. O. L. Norman Nelson, internal medicine specialist, had been in the Navy since October, 1942, as a lieutenant commander. His offices are in the Medical Arts Building.

Dr. Everett C. Perlman, pediatrician, has reopened offices in the Medical Arts Building. A lieutenant colonel in the Army, Dr. Perlman was in service from March, 1941.

Dr. Robert O. Quello has returned to general practice at 4103 Chicago Avenue. He was in service for three years and was stationed at the Army flying school in Waco, Texas, and Pampa, Texas.

Dr. Theodore L. Stebbins, served in the Army at Fitzsimons General Hospital, Denver, Colorado, and Gen-

eral Hospitals 188 and 156 in England. Dr. Stebbins is an urologist, with offices at 1409 Willow Street.

Dr. John D. Galloway, orthopedist, a major in the Army, was with General Hospital 26 in England, North Africa and Italy. He has re-opened his office in the La Salle Building.

Dr. Raymond P. Hawkinson has returned to general practice at 1025 Broadway. Dr. Hawkinson, with the rank of major, was executive officer at Hoff General Hospital, Santa Barbara, California.

Dr. Maynard C. Nelson, surgeon, a lieutenant commander in the Navy, served in the Pacific Theatre for twenty-two months, and as chief medical officer of Navy V5 at the University of Minnesota. His offices are at 421 Medical Arts Building.

Dr. Lewis J. Roberts has resumed general practice in his offices at 3827 Central Avenue in association with Dr. Hoff D. Good. A lieutenant commander in the Navy, he served twenty months with Naval Aviation in Hawaii.

Dr. Stanley W. Roberts has resumed practice in his offices at 1110 Lowry Avenue. Dr. Roberts, a lieutenant commander in the Navy, served a year with the amphibious forces in the Southwest Pacific. Later he was in the office of Naval Officer Procurement.

Dr. Arthur N. Russeth has re-opened his offices at 302 Physicians and Surgeons Building. A lieutenant commander in the Navy, Dr. Russeth was in the South Pacific with the Marines for twenty months. Later he was at the Minneapolis Naval Air Station and the Naval Hospital at Corvallis, Oregon.

Dr. Donald B. Simonson has resumed his practice at 4151 Thomas Avenue North. Dr. Simonson, a captain in the Army, served with a hospital train in France and Germany, and later at the Torney General Hospital at Palm Springs, California.

* * *

Dr. Burton Rosenholtz has returned to his pediatric practice with offices at 814 Lowry Medical Arts Building, Saint Paul, following discharge from the Navy. He served as Lieutenant Commander in the Far Pacific, much of the time caring for shock casualties aboard an Ambulance Hospital ship.

UNIVERSITY NEWS

The Board of Regents of the University of Minnesota has announced the appointment of Dr. Donald Wilson Hastings as professor and head of the Department of Neuropsychiatry in the Medical School. Dr. Hastings is replacing Dr. J. Charnley McKinley, who has resigned because of ill health.

Since his release from the Army in August, 1945, where he had served as chief psychiatrist of the Eighth Air Force in England, and later as chief Air Force Psychiatrist in Washington, Dr. Hastings has been professor of psychiatry at the Women's Medical College in Philadelphia. He is a graduate of the University of Wisconsin Medical School and interned at the Philadelphia General Hospital. He held a Rockefeller Fellowship in Psychiatry at the Pennsylvania Hospital and Institute for Nervous and Mental Diseases in 1936-38. From 1938 to 1939 he was psychiatrist at the Student's Health Service at Harvard University. Appointed clinical director of the Pennsylvania Hospital in 1939, Dr. Hastings remained there until his induction into military service. During this same period he was also instructor in psychiatry at Jefferson Medical College. He assumed his duties at the University of Minnesota on March 16.

* * *

Dean Harold S. Diehl announces the appointment of Dr. Robert A. Aldrich and Dr. Clifford G. Grulee, Jr., to special teaching assistantships in Pediatrics, and Dr. Charles U. Culmer to a similar post in Surgery. Dr. Aldrich holds the Bachelor of Arts degree from Amherst College and the Doctor of Medicine degree from Northwestern University Medical School. Dr. Grulee holds the Bachelor of Arts degree from Wayne University and the Doctor of Medicine degree from Northwestern University Medical School. Dr. Culmer holds the Doctor of Medicine and the Doctor of Philosophy degrees from Northwestern University. The funds for the support of these special assistantships have been provided by the Rockefeller Foundation as part of its program to aid in the development of selected young men whose preparation for teaching and research posts was interrupted by military service. Additional appointments are under consideration in Surgery, in Neuropsychiatry, and in Preventive Medicine and Public Health.

FACIAL INJURIES

(Continued from Page 239)

it aids in the acts of swallowing and coughing. The hemoglobin should be brought rapidly to normal by means of repeated transfusions in all cases in which the viability of important bone fragments or of large soft tissue flaps is in question. Oxygen can be administered through a nasal catheter or tracheotomy tube during the first postoperative days for the same reason. Even in severe facial injuries the patients should be out of bed for intervals on the first or second postoperative day if possible. Every effort should be made to prevent stasis and atelectasis. The postoperative use of penicillin and sulfadiazine undoubtedly helps prevent pulmonary complications.

When the jaws are immobilized a liquid diet becomes necessary. A high caloric liquid diet with supplemental vitamins is given. It is well to chart the patient's weight so that any undue loss can be noted. As a general rule, the patients lose weight the first week and thereafter maintain that weight or gain to their previous level.

Severe facial injuries are a psychiatric as well as a surgical problem. Since the patients are made ambulant as early as possible they are encouraged to circulate about the wards. They must not be allowed to develop seclusive trends.

CANCER DETECTION CENTERS

(Continued from Page 269)

feasible, clinics financed and aided by the Minnesota Cancer Society, will be extended to other communities as initiation, organization, and approval by the County Medical Societies is forthcoming. The Committee wishes to make it clear that at any time a group of physicians decides to establish a clinic, without the financial help of the Minnesota Cancer Society, it is perfectly free to do so provided it has the approval of the local County Society.

An interesting observation was the experience noted in Philadelphia[6] where over 70 per cent of the 2,552 patients examined in one year in the detection clinics were referred to their family physicians for some physical defect or other, not necessarily neoplastic in nature. Some of these clinics were originally established on the basis of one evening clinic a week, and grew with experience and demand. The experience of cancer detection clinics in Chicago[7], Milwaukee[2], New York[3], Philadelphia[4], and Los Angeles[5], testify to the value of periodic examinations of apparently healthy adults for cancer or its precursors.

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Classified Advertising

FOR SALE OR RENT—Practice and home with office and ten-bed hospital (equipped) in one unit. Located in southwestern Minnesota. Will rent for cash or on a percentage basis. Address Dr. P. C. Cress, Ellsworth, Minnesota.

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FOR SALE—General Electric Portable X-ray Model F with case, stand and 8"x10" hand fluoroscope. Just returned from factory in A-1 condition. $425.00. Address D-156, care of MINNESOTA MEDICINE.

FOR SALE—X-ray table, $10.00; suction pump (excellent condition), $30.00; microscope, $100.00; 2 infrared lamps $10.00 each; eight-bulb cradle lamp, $7.50; centrifuge, $3.00; Hyfrecator, $15.00; Baumomometer, $15.00. Address Bernice Bates, 16 N. 4th Street, Minneapolis, or telephone Main 7974.

FOR SALE—Precision medical x-ray, fluoroscope, cassettes. In excellent condition. Diathermy machine. $500.00 for all. Address D-154, care MINNESOTA MEDICINE.

POSITION WANTED—As receptionist and office nurse in physician's office. Extensive experience in supervisory work—medical, surgical, pediatric, industrial. Address Catherine Smith, 2108 Sargent Avenue, Saint Paul 5, Minnesota. Telephone: Emerson 8757.

AVAILABLE AT ONCE—Office suite for eye, ear, nose and throat specialist in new Medical Center, in desirable, high-class residential section, Minneapolis. Address D-157, care MINNESOTA MEDICINE.

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FOR SALE—Complete office equipment and new modern home in prosperous Central Minnesota small town. Unopposed. Excellent opportunity. Address D-153, care MINNESOTA MEDICINE.

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(Continued from Page 234)

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